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THE MESENCEPHALIC ROOT OF THE FIFTH NERVE.

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I.—Current Views as to Axones comprising the *Tractus Mesencephalicus Quinti* being Afferent or Efferent, or Centrifugal or Centripetal.

The origin and distribution of the mesencephalic root of the fifth nerve were very variously described until the last decade, during which a great deal of work by numerous observers was accumulated, consisting

almost entirely of investigations on the structure and destination of the axones of the large globular cells which are scattered along this root.[1] The most generally accepted description of the characteristic cells and their large axones is that given by Kölliker [21] and Ramon y Cajal [27]—namely, that the axones of the cells consist of a thick limb and of a fine collateral, or slender limb. Of these, the former descends as far as the motor nucleus of the fifth nerve, and there gives off collaterals which ramify around the cells of that nucleus. After passing this nucleus, the axones were followed by Boyce [3], van Gehuchten [33], Kölliker, &c., as far as the exit of the fifth nerve from the pons. No observer, however, has attempted to discover the destination of these fibres by tracing their course when degenerated after their division in the mesencephalon. It was the object of the present communication to answer this question as far as possible, and the material collected has also furnished the opportunity of clearing up various points concerning the central relations of the fifth nerve.

Probst (*vide infra*) described a continuation of these axones in the dorsum of the bulb, sending collaterals to Deiter's nucleus, and ending in the nuclei of the vagus and glossopharyngeal and the fasciculus solitarius. These fibres do not arise, however, from the fifth nerve (*vide infra*).

An attempt was made by Terterjanz [31] to support certain theoretical views of Kölliker on the peripheral distribution of the mesencephalic trigeminal fibres. To this end he removed the palatal muscles in a few animals, and described retrograde degeneration, as evidenced by Marchi's method, in the mesencephalic root of the fifth nerve. Examination of his drawings, however, does not, in our opinion, confirm the conclusions he founded upon them.

With respect to the fine collateral, or slender limb, of the axone of the mesencephalic cells mentioned above, no investigations of its distribution have been made, beyond the description given by Ramon y Cajal [28]. This is clearly a point for further consideration, and reference to it is made below.

A different question has now to be considered with regard to the mesencephalic root—namely, whether it has any afferent function in addition to the efferent or motor one just described. The view that it is essentially a sensory mechanism has recently been revived by

[1] Morphologically no confusion is now possible between the globular trigeminal nerve cells, which, as Obersteiner showed, are of common type in all animals, and the tectal cells lying among them and forming the nucleus intertrigeminalis of Kohnstamm. (See fig. 1.)

Johnston [15] on the basis of the study of fibre-stained serial sections of normal brains, adult and embryonic. From these he deduced the opinion that the mesencephalic fibres, after passing the motor nucleus of the fifth, entered its sensory root. He therefore regards the whole tract as an afferent mechanism. We reserve to the end of our paper the final discussion of this important question. His view, which relates only to centrifugal axones, the function of which is still indeterminate, would have gained strength had he employed the Marchi method and recognized that the root contains both centrifugal and centripetal fibres. Of the afferent character of the latter there can be no doubt.

We think it best to present the results of our work according as they illustrate the course and relation of centrifugal and centripetal axones respectively. We have investigated these both by chromatolysis and by Marchi staining of sheath degenerations.

At the same time, inasmuch as a great deal of our material throws light on the course taken by the centripetal fibres of the fifth nerve other than those running in the mesencephalic root, we add the discussion of this part of the subject also.

(II)—Examination of Centrifugal Axones in the Mesencephalic Root.

(a) By Chromatolysis.

We have endeavoured to investigate the destination of the fibres of the mesencephalic tract by an examination of the chromatolysis resulting from lesions at different points in the course of the fifth nerve. The lesions were:—

(*α*) Division of the mesencephalic root intracerebrally at different points: (1) opposite the interval between the colliculi, (2) opposite the middle of the posterior colliculus, or (3) close behind the posterior colliculus, and just ventral to the fourth nerve.

(*β*) Division of fifth-nerve roots between exit from the pons and the Gasserian ganglion.

(*γ*) Division of the first and second parts of the fifth nerve, distally to the Gasserian ganglion.

(*δ*) Division of third part of the fifth nerve distally to the Gasserian ganglion, (1) at foramen ovale, (2) by avulsion of the branches more peripherally.

Methods Employed.

For this work cats and monkeys were employed. The procedure was the same throughout. The operation of division of the mesencephalon, or of the nerves, was performed under chloroform or ether anæsthesia, and with strict antiseptic precautions. The animals were kept alive for the usual period of two to three weeks, then, after being tested physiologically, they were killed under an anæsthetic, and the arterial system perfused with 10-per-cent. solution of formalin in normal saline. The central nervous system was then removed to a similar solution, in which it was allowed to remain for twenty-four hours. The tissues were then treated in the usual way, cut in paraffin, and stained either by Unna's polychrome blue or by Pappenheim's pyronin method.

Results.

(α) *Lesions of the mesencephalic tract*, anterior or cephalad to the motor nucleus of the fifth.

The results obtained were very striking. The chromatolytic changes that occurred on section of the mesencephalic root were rapid in development and extreme in degree. In all the animals examined (four), the characteristic mesencephalic cells above the lesion had, for the most part, entirely disappeared. This notable result may best be accounted for by our view that the tract contains both afferent and efferent fibres. It is possible also that the effect of the intracerebral section is exaggerated by concurrent interference with the circulation. That the latter is, however, a very limited factor is shown by the fact that in several cases the incision was an extremely small puncture, unaccompanied by any obvious consequent thrombosis (*cf.* Kohnstamm [20]) on reactive atrophy.

(β) *Section of the roots of the fifth nerve between the pons varolii and the Gasserian ganglion.*

This operation of division of the roots of the fifth nerve between the Gasserian ganglion and the pons was performed in four animals—three cats and one monkey (*Macacus rhesus*). It was followed in every case by extensive chromatolysis of the characteristic globular trigeminal cells as previously described by Kure [30]. This particular operation furnished also the answer to an important question, which can suitably be disposed of at this point—namely, the true relation of the cells of the locus cæruleus to the fifth-nerve tracts. This includes another question, whether the multipolar cells of the locus cæruleus are in any way part

of the trigeminal system. Kohnstamm and Quensel [18] regard them as being part of a nuclear system, in relation with the globular cells and the coarse axones of the mesencephalic root. Evidence for this view, however, is not obtained from our experimental material.

Personally, we have seen nothing to suggest that these cells undergo change either of a direct kind, when the afferent trigeminal channels are interrupted, or of a retrograde type, when the mesencephalic trunk has been divided at a sufficient distance from the locus cæruleus to avoid actual involvement of it in the injury.

Further, the photograph (fig. 12) of a section through the locus in a patient in whom the Gasserian ganglion had been removed six weeks before demonstrates that the cells of the ipsilateral nucleus are unaffected.

Our conclusion, therefore, is in confirmation of that of Cajal, that these cells constitute a separate ascending system, which is not connected, at least directly, with the fifth nerve.

(γ) *Section of divisions of the trigeminal nerve, distal to the ganglion.* In the monkey (*Macacus rhesus*) it was found that section of the superior and middle divisions of the trigeminal nerve, just peripheral to the Gasserian ganglion, produced no signs of chromatolysis in the globular cells of the mesencephalic root or in those of the locus cæruleus. The completeness of the lesion was confirmed by the presence of total degeneration of the branches of these two divisions, as shown by Marchi staining. It seems reasonable to conclude, therefore, that the centrifugal axones of the mesencephalic root are not continued into either of these divisions of the nerve.

(δ) Very different are the results of *section of the inferior division*, or avulsion of its branches. Intracranial section of this division at the foramen ovale was performed on three animals (one cat and two monkeys). Extracranial avulsion of its branches in the spheno-maxillary fossa was done in four cats, while in one the motor branches were removed, leaving the sensory part intact. In all these animals chromatolytic changes were observed in many of the characteristic mesencephalic root cells throughout the entire length of the root. The typical changes (see figs. 1 and 2) were exhibited—namely, the extrusion of the nucleus to one side and the fusion of the chromatin granules—but what was especially striking was the fact that, even in so short a time as twenty-one days, some of the cells had shrunk to mere remnants or had almost entirely disappeared.

In the animal (cat) in which the motor branches were destroyed without injury to the sensory branches, the chromatolysis was also

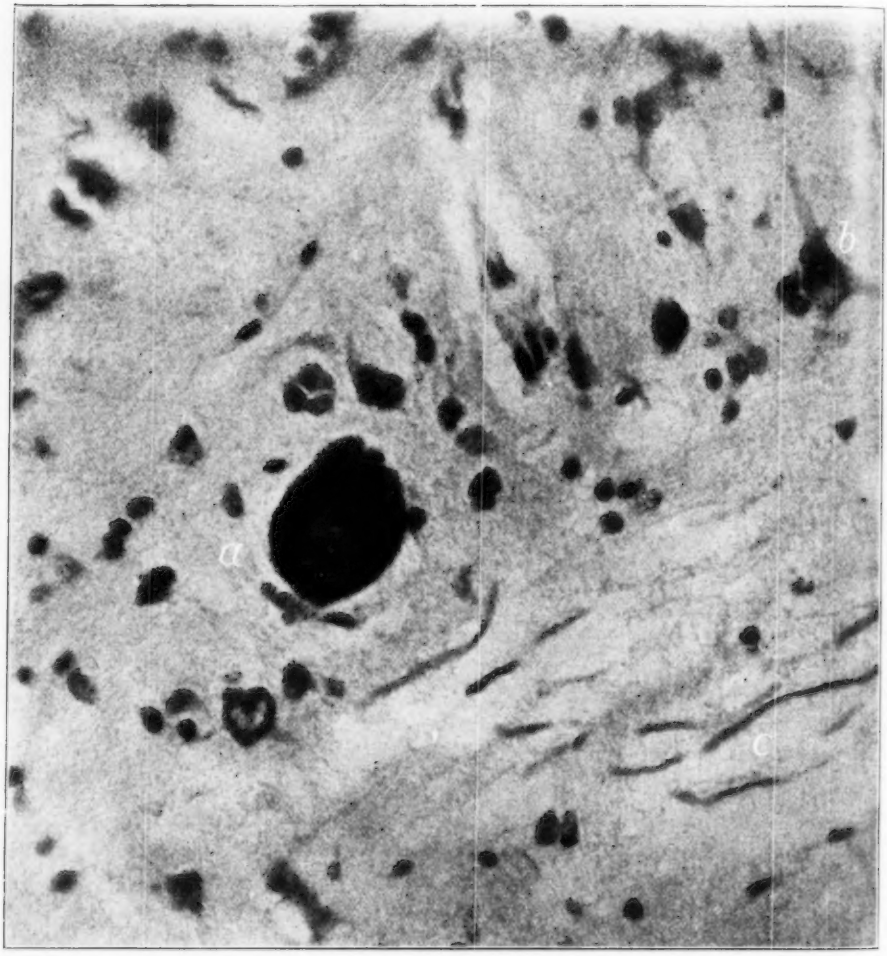

FIG. 1.

Macacus rhesus—Normal. Tractus mesencephalicus quinti—showing typical globular nerve corpuscles (*a*) of the tract, as well as axones; also a cell of the nucleus intratrigeminalis (*b*) of Kohnstamm, and axones of the tractus (*c*).

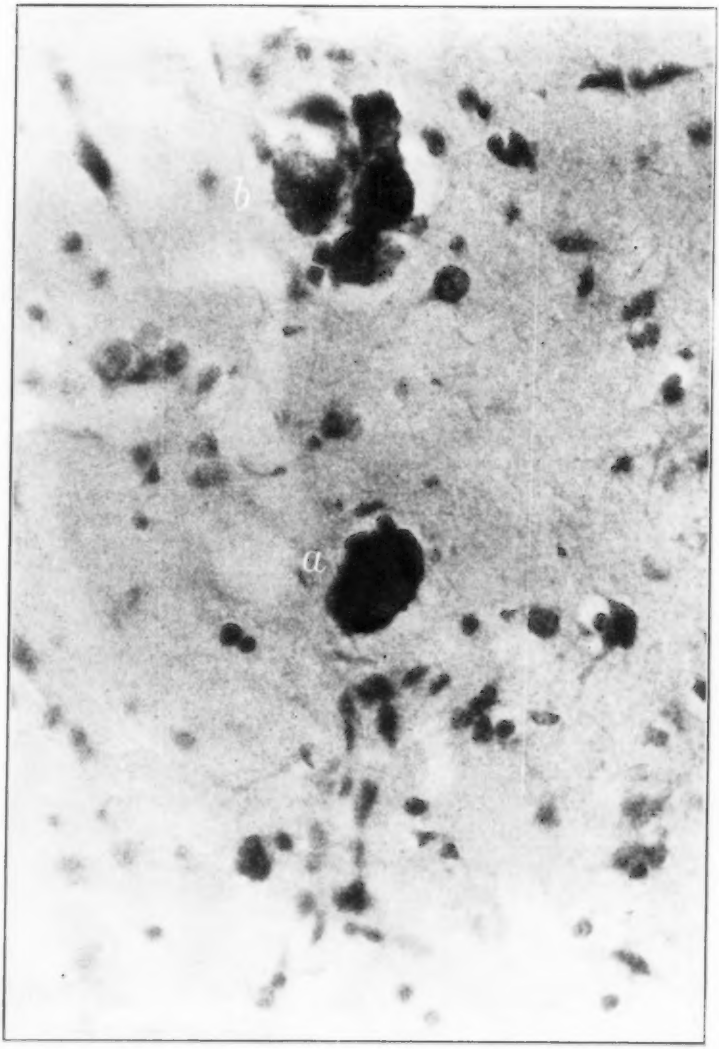

FIG. 2.

Macacus rhesus.—Chromatolysis of the globular cells of the tractus mesencephalicus quinti after avulsion of the inferior division of the fifth nerve. The solitary cell (*a*) shows shrinkage, lateral position of the nucleus. The group of cells shows (*b*) complete destruction of the cells and disappearance of the nuclei.

present. This observation does not lend support to Johnston's view that the axones enter the sensory root.

Just as in the division of the nerve proximal to the ganglion, so in these experiments of division distally to it there was no change in the cells of the locus cæruleus, although the cells in the mesencephalic root showed marked alteration.

Finally, these series of experiments showed particularly clearly the truth of the principle enunciated by Gudden and Forel and others, that the degree of chromatolysis depends upon the site of the injury. Thus the degree and rapidity of the destruction of the nerve cells increases as we proceed with the experimental sections in the following order :—

(*a*) Section of peripheral branches distal to the root ganglion.

(*b*) Section of the roots of the trigeminus between the root ganglion and the pons varolii.

(*c*) Section of the trigeminal roots in the pons varolii.

(*d*) Section of the tractus mesencephalicus trigemini in the plane of the posterior colliculus.

The last-named lesion caused, as stated above, total disappearance of the nerve cells in a very short time.

(*b*) *By Degeneration of Centrifugal Axones of the Mesencephalic Root following its Intra-cerebral Division.*

Operative Procedure.

To produce centrifugal degeneration in the mesencephalic root alone we divided it by exposing the mesencephalon at the level of the anterior border of the tentorium, and then making a small puncture incision—(*a*) through the side of the posterior colliculus,[1] or (*b*) just in front of it, or (*c*) just behind it where the fourth nerve passes out. We thus obtained section of the mesencephalic root at three points in its course, the lowest of which was at a considerable distance above the motor or masticatory nucleus.

We hoped by these measures to determine completely the ultimate destination of the mesencephalic root axones. To this end we employed the Marchi method. The Gasserian ganglion and the peripheral branches of the nerve were also stained by the same method, the latter being first dissected out and separately identified. After staining, these

[1] Corpus quadrigeminum posterius.

nerves were examined for degenerated fibres by teasing. The ganglia were cut in serial section for the same purpose.

In our first series of experiments the results seemed so contrary to expectation that we thought there must be some underlying error of observation. Accordingly we extended the number of experiment until on this point our series included twenty monkeys and ten cats.

Results Obtained.

In the first place, the amount of degeneration of the centrifugal axones is exactly proportional to the number of cells above the site of the lesion. This is the converse proof of the statement already made in respect of the chromatolytic results which follow intracerebral division of the mesencephalic root (*vide* p. 179)—namely, total destruction and disappearance of the globular cells situated above the lesion.

In the second place, the course taken by these axones is that described by Boyce, Cajal, and others—namely, curving round the outer side of the motor nucleus, sending collaterals thereto, and passing on, to make their exit from the pons between the sensory and motor roots (see figs. 3 and 4). As the accompanying photograph shows, these fibres in the monkey (*Macacus rhesus*) traverse the sensory root in part, but ultimately approach the motor root. Comparison of our photographs with the figures drawn by Johnston [15] of the path of these fibres in the human embryo shows that the path taken by the axones is the same in different species of the Primates; but, in contradistinction to the conclusions arrived at by him, we consider that their ultimate relation is with the motor and not the sensory root (see fig. 4). On this question the degeneration method affords irrefragable evidence.

We have now to consider three points in connexion with the axones during their course through the side of the pons: (1) in the first place, the question of axones, or principal collaterals from them, passing down into the bulb, as described by Probst; (2) in the second the number and degree of collateral supply to the motor nucleus; and (3) in the third, the continuation of the axones from the pons.

(1) The statement made by Probst [27] that some of these axones run caudally, sending collaterals to Deiter's nucleus, and terminating in the nuclei of the vagus and glosso-pharyngeal, and the fasciculus solitarius, is, in our opinion, not borne out by the facts, when the lesion is limited to a simple division of the mesencephalic root. In his experiments, the lesion made with a hook and cannula was extensive, and

FIG. 3.

Cat.—Section.—Showing how the fibres of the tractus mesencephalicus (*a*) pass outwards through and among the fibres of the sensory root (*b*) (but gain ultimately the motor root).

In the experiment, which was one of a mesencephalic lesion, Monakow's rubrospinal tract was injured, and is shown (*m*) degenerated in transverse section, just to the inner side of the fifth nerve.

FIG 4.

Macacus rhesus.—Section through exit of V. The mesencephalic root had been divided intracerebrally, and the degenerated fibres can be seen leaving the pons with the *motor* root of the trigeminal.

involved the fibres of the fastigio-brachial system (Clarke and Horsley), from which bulbar collaterals proceed as well as other fibre systems. We have thoroughly examined the large mass of material at our disposal, and are certain that no collaterals proceed from the axones of the mesencephalic root to any nucleus lower than the oral plane of the vestibular nuclei.

Probst's view is repeated in another form by Lewandowsky [22] who, while opposing Probst's statements as to the termination of the supposed fifth-nerve collaterals, nevertheless ascribes to a descending bundle passing dorsally over the seventh nerve and ending ventrally of the nucleus of the solitary bundle an origin from the fifth-root region, and possibly from Kohnstamm's nucleus intratrigeminalis.

We have no doubt that the fibres observed by Probst and Lewandowsky are two different tracts, neither of which arises from the fifth roots. The first of these [see figs. 5 (*b*) and 6 (*b*)] consists of fine fibres, which by the lesion are caused to undergo retrograde degeneration backwards into the "gustatory nucleus" of Nageotte. Thus the section of the mesencephalon divides these neurones of the second order as they ascend from the nucleus of Nageotte to the thalamus. Hence, not only can they be traced by retrograde degeneration into the gustatory nucleus, but also upwards into the thalamus. They pass upwards in the reticular substance between Forel's bundle and the central grey matter lying dorsal and external to the remains of the posterior longitudinal fasciculus. Higher, the fibres pass into the lamina interna, and gradually into the inner part of the dorsal third of the nucleus lateralis.

The second bundle of coarser fibres described by Lewandowsky are internal, and, as he states, ventral in position to the grey matter surrounding the fasciculus solitarius [see figs. 5 (*a*) and 7 (*a*)]. These are, in fact, some of the most dorsal bundles of the substantia reticularis bulbaris, and terminate before the central plane of the hypoglossal nucleus is reached.

We have no evidence of the precise origin of these coarse reticular fibres of Lewandowsky, which must arise from cells at or above the level of the superior colliculi. They are efferent or descending fibres, as they do not degenerate upwards.

That these two tracts of fibres, Probst's and Lewandowsky's, have nothing to do with the fifth nerve is amply proved by our experiments, since we have in one series examples of all possible variations of the experimental conditions—i.e., complete section of the fifth mesencephalic

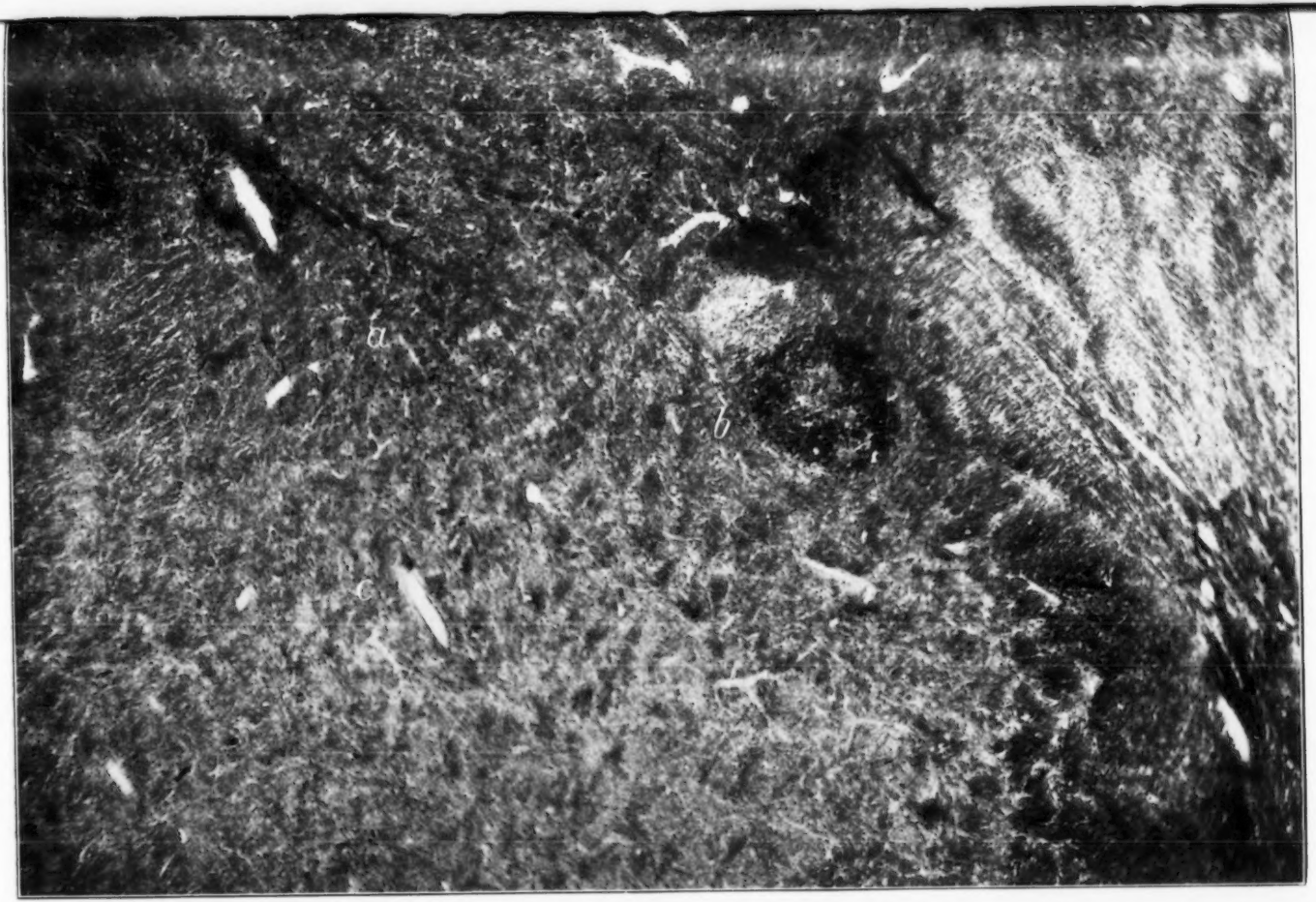

FIG. 5.

Macacus rhesus, 204·09.—Section of mesencephalon caudal to fig. 4, and showing both tracts ascribed by Lewandowsky and Probst erroneously to the fifth nerve.

The coarse-fibred tract (*a*) in the reticular substance is Lewandowsky's; the fine retrograde gustatory fibres in the fasciculus solitarius (*b*) are those

FIG. 6.

Macacus rhesus, 204·09.—Section of mesencephalon parallel to the interaural plane (Clarke and Horsley) in case of division of the tractus mesencephalicus.

The coarse degenerated fibres of the latter are seen at (*a*) descending to leave by the motor root. Outside these, and separated from them, are the fine retrograded gustatory fibres (*b*).

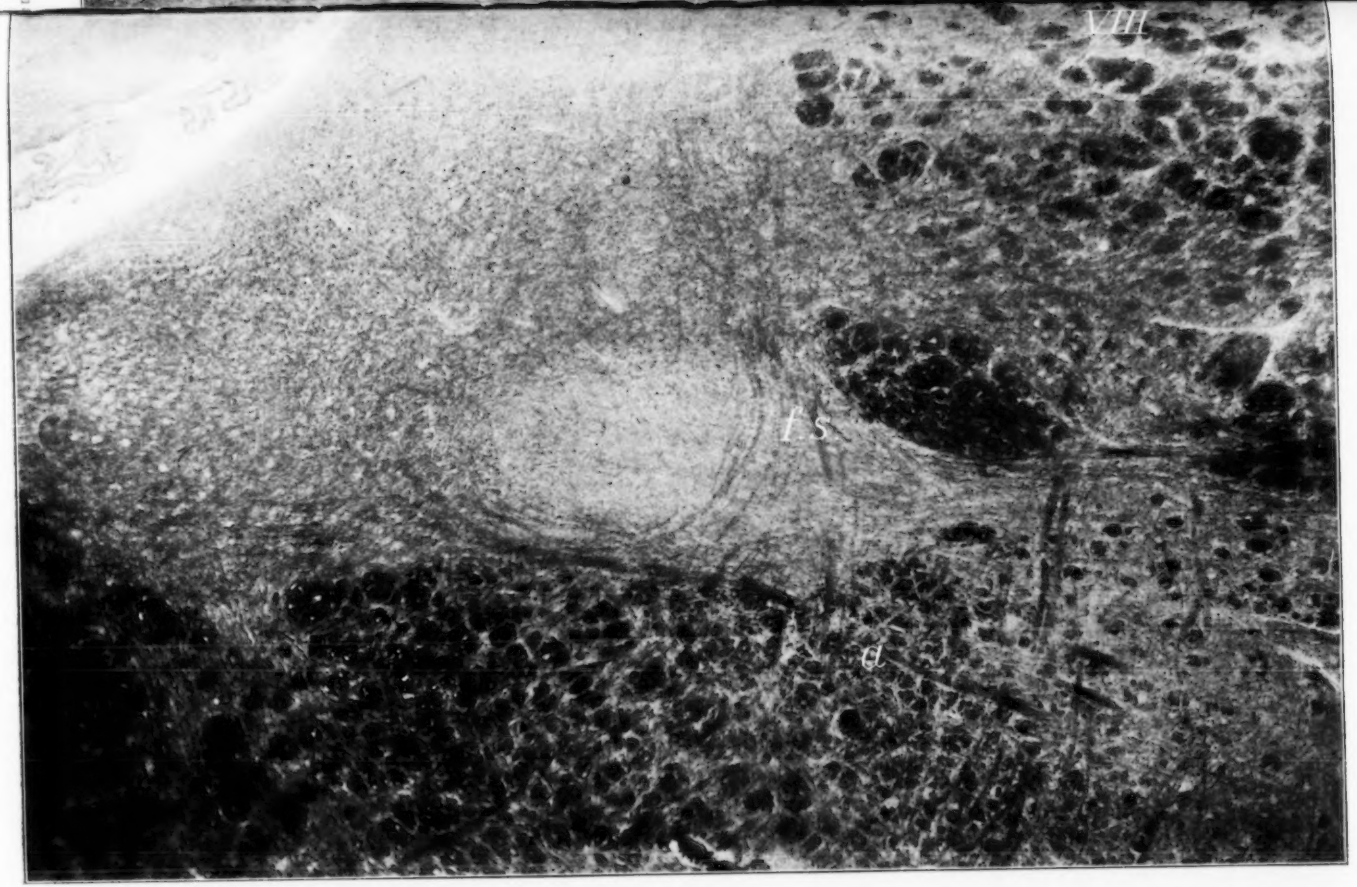

FIG. 7.

Cat.—Section of the vagal angle and lateral wall of the fourth ventricle (*IV*), showing the fasciculus solitarius (*fs*) and its nucleus. Below its nucleus are (*a*) the coarse degenerated fibres of Lewandowsky, occupying the upper and outer border of the pars reticularis. These fibres come from the neighbourhood of a mesencephalic lesion.

It will be observed that they have no connexion with the fifth nerve, the mesencephalic root of which was completely divided. *IV* = the fourth ventricle. *VIII* = the descending root of the eighth nerve.

root without either of these tracts being degenerated, section of the root with both or only one tract degenerated, and degeneration of both tracts without section of the mesencephalic root of the fifth nerve.

In short, we have all possible control experiments, from which it is plain that neither the fine-fibred tract of Probst nor the larger-fibred reticular tract of Lewandowsky is formed by collaterals from the mesencephalic root of the trigeminus.

(2) *Supply of Collaterals to Motor Nucleus.*—The supply of collaterals from the mesencephalic root, which Cajal was the first to describe, is, of course, well demonstrated in Marchi preparations [see figs. 8 (rhesus) and 9 (man)]. All the collaterals are very fine, and form a network which stains intensely by this method.

(3) *The Continuation of the Axones from the Pons.*—We now approach what was originally the starting-point of the present investigation—namely, the destination of the centrifugal axones of the mesencephalic root after their exit from the pons. Physically, this can be investigated only in the manner we have used—namely, intracerebral section of the mesencephalic root, with subsequent examination of the degenerated fibres.

In addition, we examined, by the teasing method, the various nerves distributed to the ocular muscles, the sixth, fourth, and third, including the ciliary branches. In several experiments, considerable degeneration was found in the fourth nerve, but this was in every case the result of damage to that nerve in the course of the operation on the mesencephalon. The other nerves in the orbit were entirely free from degeneration, a result which confirms the observations of Kohnstamm and Quensel, who failed to find any chromatolysis of the characteristic cells of the mesencephalic root after removal of the orbital contents in animals.

As has already been indicated, Marchi preparations of the trigeminal nerve and its branches, from animals in which intracerebral section of this root had been performed, failed to demonstrate these fibres in any part of the trigeminal distribution peripheral to the Gasserian ganglion. They could be seen in the motor root as far as the level of this ganglion, but could not be found by teasing or by serial sections in the various peripheral branches.

In view of the fact that the conditions under which intracerebral degeneration develops are not precisely the same as those governing the degenerative changes in the peripheral nerves, it occurred to us that we ought to re-investigate the simple question of the degeneration of the

FIG. 8.

Macacus rhesus, 11·07.—Complete degeneration of tractus mesencephalicus quinti, showing the collaterals refrom passing into the motor nucleus (*m m*). The latter nucleus lying between the letters *m m* is marked off by son of the number of fine collaterals (degenerated) in it.

Above, at *g*, are seen the fine "gustatory" fibres passing backwards and outwards.

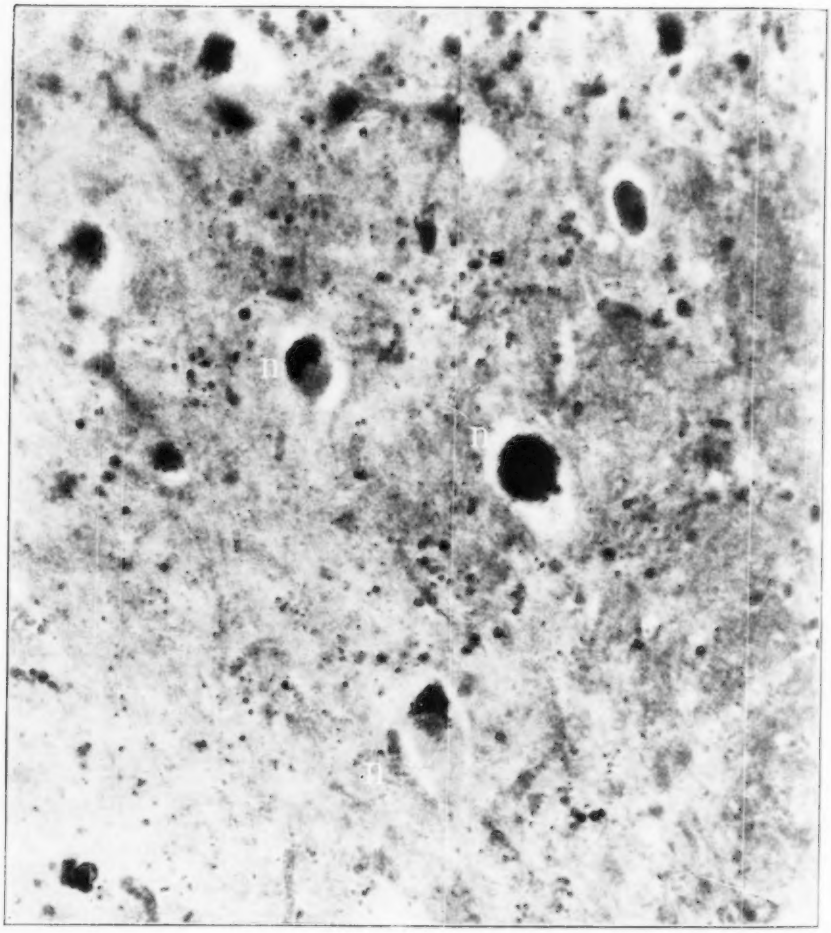

FIG. 9.

Man.—Collaterals (retrograde degeneration) in the motor nucleus of the fifth nerve consequent upon removal of the nerve six weeks before death. The nerve-cells (*n n*) contain much pigment, staining deeply with the osmic acid.

fibres of an efferent motor-nerve when they are divided from their nucleus of origin while still within the central nervous axis. To test this by a simple case, we made three experiments on the hypoglossal nerve—two in the cat and one in the monkey. The experiment was as follows: In the anæsthetized animal the cerebellum and bulb were both carefully exposed, a small knife was then introduced, parallel to the plane of the fourth ventricle, through the tubercle of Rolando. The direction given to the knife was such that the hypoglossal fibres running through the tissues of the bulb were divided close to their nucleus. The animals were killed fourteen days after the operation. In each case the peripheral nerve showed a proportionate number of completely degenerated fibres. It is clear that a simple efferent nerve, like the hypoglossal, degenerated completely when its axones are divided in their intracerebral course. It seemed to us that the apparent anomalous fact of the mesencephalic trigeminal fibres was susceptible of three different explanations: (*a*) That the fibres are distributed to some other structure—e.g., the otic ganglion. (*b*) That they lose their medulla, and are continued to the periphery as non-medullated fibres. (*c*) That they end in the Gasserian ganglion.

In order to test the first assumption, a further series of experiments (three in number) were made in which, after a mesencephalic lesion had been made as above stated, the Gasserian ganglion was removed *en bloc* with the contents of the spheno-maxillary fossa and serial sections cut. In these again the degenerated fibres could not be traced beyond the level of the Gasserian ganglion, and the otic ganglion was found to be quite free from any such fibres.

The second explanation (*b*) is rendered improbable by the fact that all the fibres of the various peripheral branches, as seen in teased specimens stained by osmic acid, appear to be medullated. It would appear then that the third explanation (*c*) must be accepted as the most probable—namely, that the efferent fibres of the mesencephalic root end in the Gasserian ganglion. At first sight this mode of termination would seem to be incompatible with the fact stated above, that some chromatolysis of the cells of origin occurs after avulsion of the peripheral branches of the inferior division of the trigeminal. But on this point Warrington [37] has shown by section of anterior and posterior roots respectively that the chromatolytic changes resulting therefrom affect not only the cells in direct association with the fibres divided, but also other cells forming part of the same nerve centre. For all these reasons we prefer to leave this matter at this point.

III.—*Examination of Centripetal Axones.*

There is abundant evidence that the mesencephalic root contains, in addition to the centrifugal fibres described above, a considerable number of centripetal fibres.

If this root is cut across at the level of the posterior corpora quadrigemina, sections of the mid-brain cephalad to the lesion show the presence therein of a varying number of fine degenerated fibres (fig. 10). The question immediately presents itself: Are these fibres derived from cells in the brain-stem, or from those of the Gasserian ganglion?

The idea that some of these may be fine collaterals resulting from the divided axones of the characteristic globular cells we owe to Van Gehuchten. Our method does not permit of expressing an opinion as to whether any of the degenerated fibres we have observed originate in this way. We have, however, the proof that many of the ascending fibres in the root come from the cells of the Gasserian ganglion. Upon this question, which has received direct attention from van Londen [35] and Bregmann [4], who each observed by the Marchi method degenerated fibres in the tract after section of the trigeminal proximal to the ganglion, and from Probst, who observed the same after a lesion of the mesencephalon at the level of the exit of the fourth nerve, we have accumulated a large amount of material of the following kinds:—

(*a*) Degeneration following removal of Gasserian ganglion: (*α*) Man. (*β*) Other animals.

(*b*) Intracerebral section of the mesencephalic root: (*α*) At level of exit of the fourth nerve. (*β*) At level of anterior border of posterior colliculus.

(*a*) *Degeneration following Removal of Gasserian Ganglion.*

(*α*) *Man.*

In a patient (H.) in whom the Gasserian ganglion was removed by one of us (V.H.) in 1907 at the National Hospital for the Paralysed and Epileptic, and who, shortly after her discharge from hospital, contracted a rapidly fatal pleurisy, examination of the brain by the Marchi method revealed the following—(death occurred six weeks after the operation). As shown in the photograph (fig. 11), the trunk of the nerve in its transit through the pons displayed complete degeneration—i.e., not only of the fine afferent fibres, but also a retrograde degeneration of the motor root. As the opportunity of tracing these degenerations in man are infrequent,

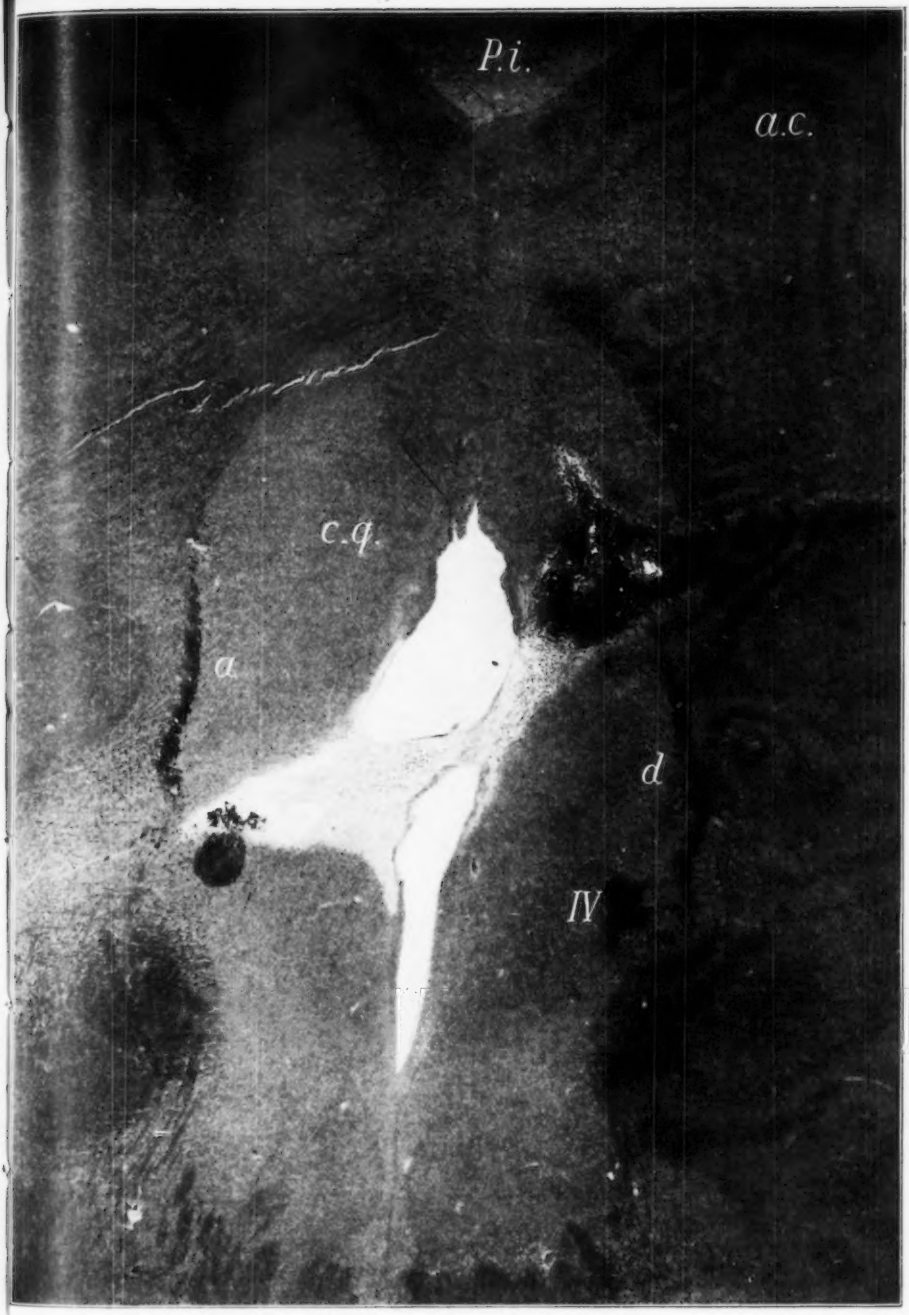

FIG. 10.

Macacus rhesus.—Section of mesencephalon through the anterior colliculi and parallel to the interaural plane.

In this experiment the knife passed across the aqueduct obliquely, thus dividing the tractus mesencephalicus quinti, on one side at the anterior region of its origin, and at the posterior (caudal) on the other. Consequently the afferent (including retrograde degeneration) and efferent fibres respectively are degenerated.

Aq = aqueduct, *l* = lesion, *c g* = central grey, *a c* = colliculus anterior, *p i* = glandular pinealis, *a* = ascending fibres, *d* = descending fibres.

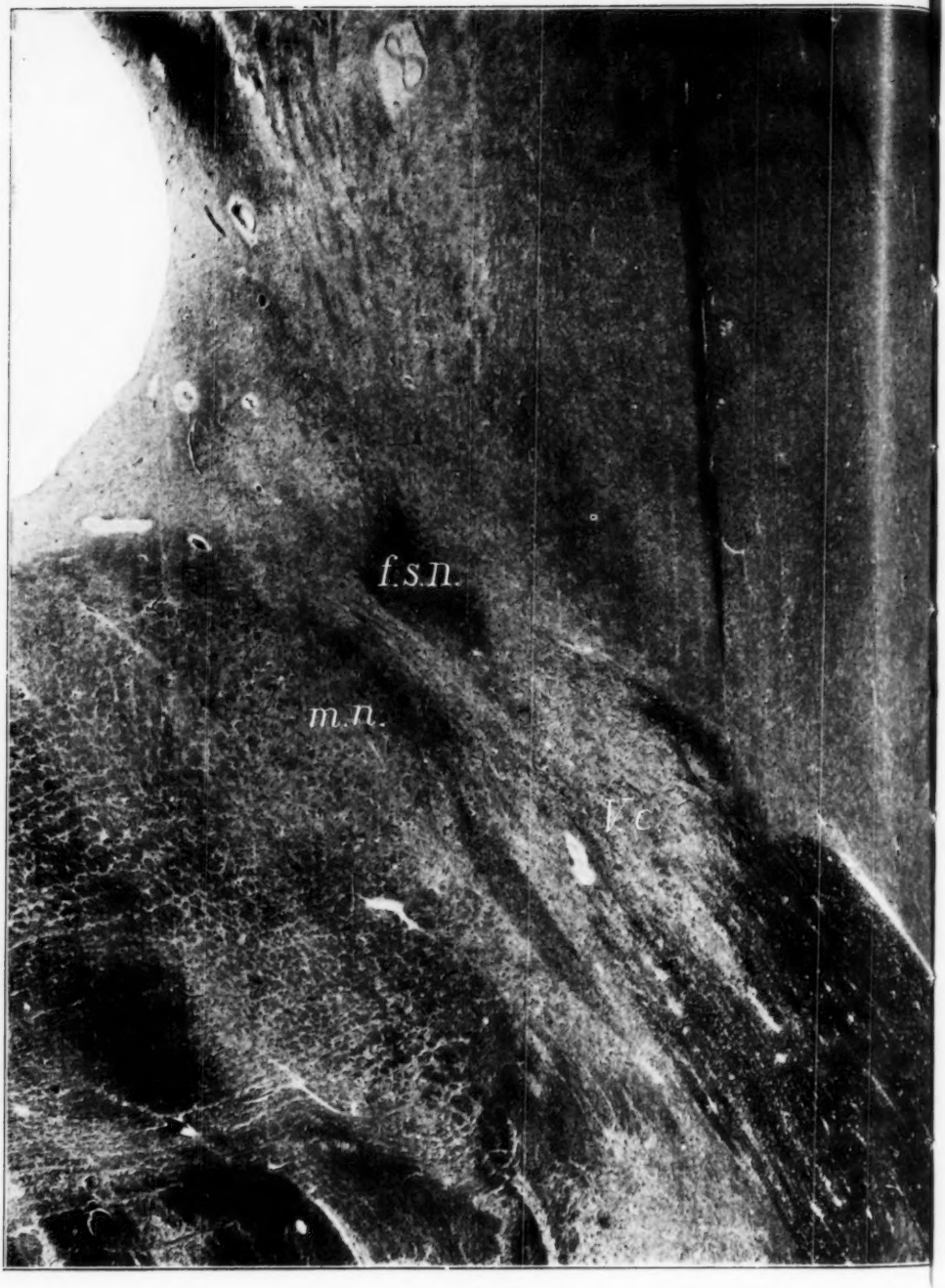

FIG. 11.

Man.—Degeneration of the fifth nerve consequent on removal of the Gasserian ganglion and roots six weeks b death, showing relation of trigeminal afferent fibres to the fasciculus solitarius.

V c = commencement of the trigeminal sensory nucleus into which enter the degenerated fibres of the nerve

m n = the motor nucleus of the fifth nerve.

f s n = fasciculus solitarius commencing from the gustatory nucleus.

we venture to add a general description to the special notes made on the mesencephalic root, and we will commence with this retrograde degeneration of the motor root.

(*a*) *Motor root* (Efferent fibres).

The retrograde degeneration of the motor fibres consists of comparatively fine granules. This may possibly be due in part to the considerable period elapsing between the operation and the time of death. The degeneration can be traced as fine collaterals in a considerable belt lying along the outer border of the motor nucleus (Ramon y Cajal's network). From this they are disseminated throughout the nucleus (see fig. 9).

(*b*) *Sensory root* (Afferent fibres).

The afferent fibres which make up the large volume of the nerve roots begin to break up into bundles as soon as they enter the outer part of the reticular substance of the pons, and at the level of the upper border of the trapezium islands of nuclear substance (convolutio quinti) begin to show themselves. Of these, one in particular, in the direct line of entry of the nerve root, and situated above and to the outer side of the dorsal end of the motor nucleus, is rendered noticeable by the particularly dense felt-work of fine fibres composing its periphery. Passing onwards, the finest fibres, or collaterals from them, turn inwards and enter the small-celled angular nucleus of the eighth nerve.

Many of the fibres go straight up to the angle of the fourth ventricle, and thence are continued as fine axones up the mesencephalic root. These are shown in detail in the accompanying photographs.

That they were not observed by the earlier investigators was probably due to imperfect staining. In their ascent in the root, they gradually come to occupy the median half of its breadth (see fig. 12 *a f*). They can be traced up as far as the central plane of the anterior colliculus, where they are seen as a single vertical row of fibres at the border of the central grey matter at its widest part. It is interesting to note that, in accord with our experimental results, the characteristic spheroidal cells are notably diminished in number and in size on the operated side. On the other hand, the cells of the locus cæruleus in this case show no difference at all on the two sides (fig. 12), an observation which also agrees with those we have made after experimental avulsion of the nerve roots in animals.

The present case afforded also a good opportunity for examining the relation of the afferent fibres of the fifth nerve to the gustatory nucleus

FIG. 12.

Man.—Transverse section of lateral angle of mesencephalon from a case of removal of the Gasserian ganglion a fifth nerve six weeks before death, showing (1) the fine afferent fibres in the mesencephalic root; (2) that these a grouped in the mesial half of the tractus as they ascend under the tectum; (3) that the cells of the locus cærule are unaffected.

l c = locus cæruleus cells deeply pigmented, but normal.

a f = placed just on the inner border of the mesial half of the tractus mesencephalicus indicates the degene tion in this of the afferent fibres.

s c p = pedunculus cerebelli uperior.

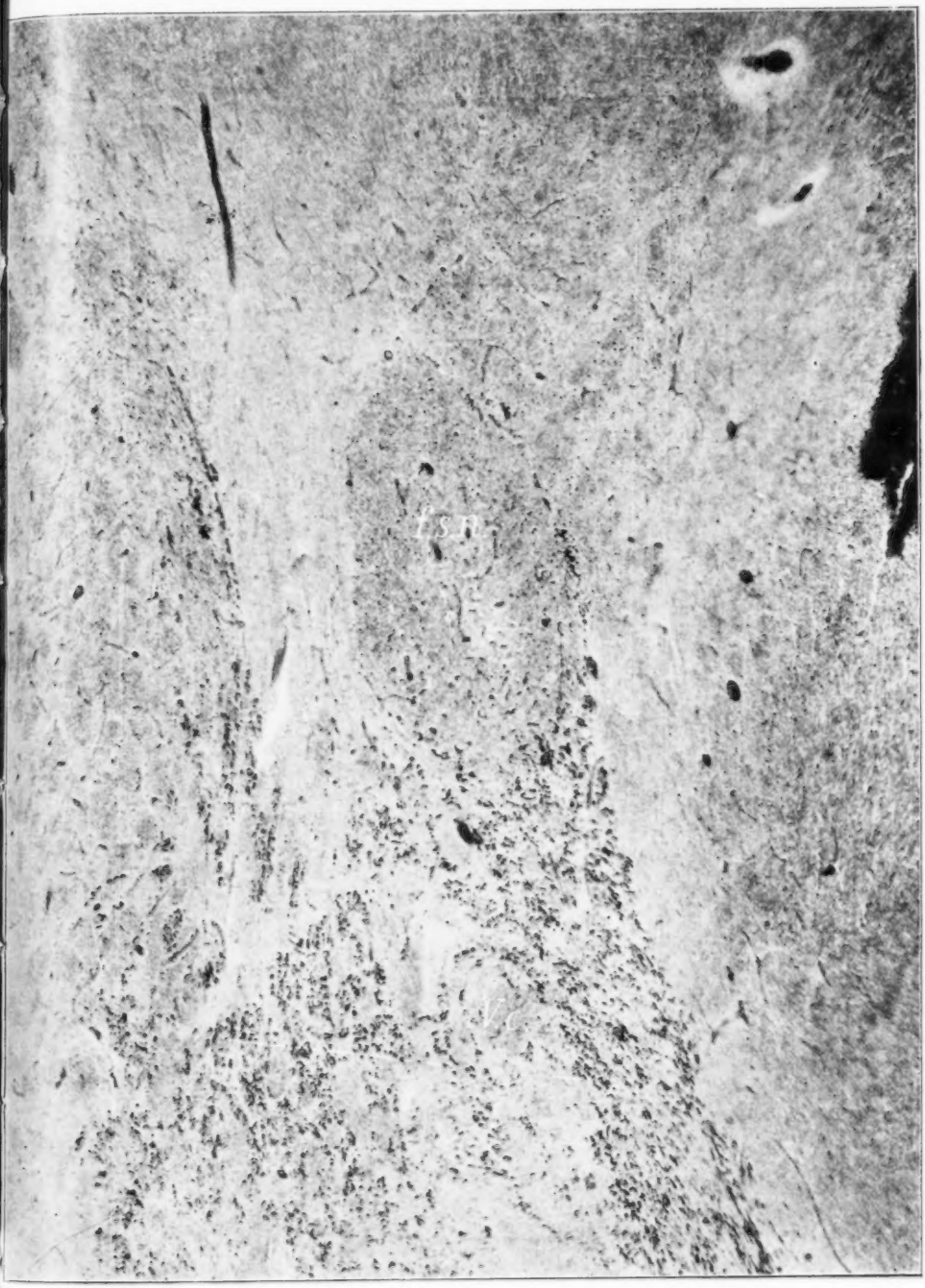

FIG. 13.

Man.—Section of the mesencephalon caudad of Fig. 11, showing the distribution of the trigeminal fibres ventral to the fasciculus solitarius.

v c = the sensory nucleus (convolutio quinti) of the fifth nerve, with numerous degenerated fibres reaching to the commencement of the fasciculus solitarius from the "gustatory" nucleus *f s n*.

of Nageotte and the fasciculus solitarius. Fig. 13 shows fibres passing into and through the outer part of this nucleus and fasciculus. A few fine collaterals can be observed in the upper part of the nucleus (*f s n*), thus demonstrating the degree of the supply of this part by the fifth nerve. This is the superior part of the gustatory nucleus of Nageotte [25].

Ventral to this, the substantia gelatinosa of the sensory nucleus of the fifth nerve extends upwards and inwards like a sharp wedge and forms the outer limit of the substantia reticularis. The fibres running through this are grouped in bundles separated by large areas of grey matter, in which they break up into fine collaterals. This portion of the fifth sensory nucleus is, lower down, sharply marked off from the grey matter surrounding the fasciculus solitarius by root fibres of the ninth nerve. The general outline of the area occupied by the nerve is, roughly speaking, a triangle, the base of which is formed by the crescentic margin which is conventionally used to represent the spinal root of the fifth nerve in most drawings of this region. This triangular outline is traceable right through the bulb to below the level of the nucleus ambiguus, caudal to which the reticular fibres certainly diminish in number, and an approach to a crescentic shape becomes more evident. Fine collaterals are supplied to the nuclei which border the nerve in every direction—namely, those of the cranial nerves from the sixth to the tenth inclusive (? other nuclei).

Crossing of Fibres (Sensory and Motor).

The crossing which the earlier writers (from Kölliker to Bruce) thought to occur, and by which fibres, both motor and sensory, of the fifth nerve of one side entered or left the roots of the opposite side, was never, in our view, justified by the facts. The nearest approach to crossing which we have seen in our specimens, either in man or the lower animals, has been merely the supply of coarse or fine collaterals to the nucleus of the sixth nerve on the same side.

This, however, is not the view taken by van Londen [35], who thought he had observed in rabbits that some of the fibres in the sensory root passed across the raphe in a plane corresponding to the level of the locus cæruleus.

(β) *Centripetal Fibres as observed in Animals other than Man after Lesions of the Nerve outside the Brain.*

In the cat and Rhesus monkey the same facts were observed as in man, with the additional interest of proving the source of the fine

afferent axones in the mesencephalic root to be the cells of the Gasserian ganglion. Thus, in two sets of animals observations were made according as the fifth nerve was divided proximally or distally to the ganglion respectively. In the former series the afferent fibres were degenerated, in the latter they were not. The degenerate axones were almost all of fine calibre, only a few of medium size being observed.

(*b*) *Degeneration of Centripetal Axones after Intracerebral Section of the Mesencephalic Root in Animals.*

In the large series of experiments in which we have made a lesion of the mesencephalon either below, at the point of exit of the fourth nerve, or at various points in the mid-brain up to the posterior margin of the anterior colliculus, we have obtained absolutely constant results. The root, under these conditions—namely, intracerebral section—contains numerous fibres running centripetally, consisting for the most part of fine axones, but including others of a medium size. The origin and course of the fine fibres we have just discussed. As regards the medium-sized fibres, it is readily conceivable that these may be stouter centrifugal axones of the globular mesencephalic cells undergoing "retrograde degeneration." At present we know of no means whereby to differentiate between these.

IV.—*Physiological Tests in Animals in which Intracerebral Division of the Mesencephalic Root was Performed.*

Our experiments on the matter of function of the mesencephalic root have been negative as far as any direct result from the lesion is concerned. In the first animals experimented upon, we investigated the sensory surfaces supplied by the fifth nerve—e.g., the conjunctivæ, skin, and nasal and buccal mucous membranes—without being able to discover that the destruction of the mesencephalic root caused any definite functional loss. Similarly there was no obvious loss of power of mastication on either side, the jaw opening without any lateral deviation. Finally, stimulation of the root on the cut surface of the mesencephalon produced no effect on the muscles of mastication, unless the excitation spreads to the pontine masticatory nucleus.

Cajal suggests that the function of the mesencephalic root is to act as a relay in connexion with stimuli emanating from the cerebral cortex, but no evidence has yet been adduced in support of this.

V.—*Summary of Results.*

(1) The mesencephalic root of the fifth nerve contains both centrifugal and centripetal fibres.

(2) The centrifugal fibres are the axones of the characteristic globular cells.

(3) Practically all these axones leave the pons by the motor root of the fifth nerve.

(4) There are some grounds for believing that some of these end in the Gasserian ganglion.

(5) But avulsion of the peripheral branches of the inferior division causes chromatolysis of the characteristic cells, a result suggesting that these axones run in the peripheral branches, though examination by the Marchi method has failed to reveal them.

(6) There is no evidence that the cells of the locus cæruleus form part of the axonal system of the trigeminal nerve.

(7) The tracts described by Probst and Lewandowsky as consisting of collaterals derived from the mesencephalic trigeminal axones, and establishing connexions with the nuclei of the eighth to eleventh cranial nerves, have no relation to the fifth-nerve system.

(8) The centripetal fibres of the mesencephalic root consist of fine axones, which are in the large majority derived from the cells of the Gasserian ganglion. Some may be derived as collaterals from the centrifugal axones (Van Gehuchten).

(9) All the evidence at our disposal is in favour of the view that the intracerebral connexions of each fifth nerve preserve a strictly ipsilateral distribution, and negatives absolutely the existence of crossed roots as postulated by earlier authors.

(10) There is no evidence that the mesencephalic root is connected in any way with the innervation of the extrinsic muscles of the eyeball or palate.

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STRANGULATION OF THE NERVI ABDUCENTES BY LATERAL BRANCHES OF THE BASILAR ARTERY IN CASES OF BRAIN TUMOUR.

With an Explanation of some Obscure Palsies on the Basis of Arterial Constriction.[1]

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Introduction.

Diplopia so often occurs in cases of intracranial tumour that one feels at times almost justified in including this symptom with the manifestations more or less commonly ascribed to the general increase of pressure—headache, choked disc, vomiting, colour inversion, extracranial venous stasis, convulsions, dizziness, bradycardia, and the like.

That oculomotor or abducens palsies may represent true "neighbourhood" symptoms in case the lesion has directly affected either the nerve centres or the nerves themselves in their intracranial course needs no comment, for the rule applies to other cerebral nerves as well as to the third and fourth pairs. It is a matter of common knowledge, however, that ocular palsies, either stationary or fluctuating in their intensity, particularly those manifested by a convergent squint, are so often misleading from the standpoint of topographical diagnosis that they have come to be classed with the "false localizing signs" of tumour.

Possibly the most complete discussion of the diagnostic confusion which may arise from this and other sources occurs in Collier's admirable paper [3]. False localizing signs, attributable in most instances to the "indirect intracranial results of intracranial new growth," were present in 12·5 per cent. of the 161 cases forming the basis of his studies. He emphasizes the fact that an "indirect" paralysis affects the abducens more often than any other cerebral nerve, and in twelve of his cases some affection of the sixth pair accompanied tumours situated above

[1] Presented at the meeting of the American Neurological Association, Washington, May 2, 1910.

the tentorium. The paralysis was unilateral in six instances and bilateral in six, and when unilateral was right-sided in four cases and left-sided in two. In two instances, furthermore, paralysis of the oculomotorius (bilateral in one case) occurred secondary to paralysis of the abducens (bilateral in the other case), the nerves in each instance being affected earlier upon the side of the lesion, which in one instance proved to be a right subcortical tempero-sphenoidal tumour, and in the other a large left prefrontal growth.

The usual explanation of these "distant" abducens lesions is based on the presumption that the sixth nerves, owing to their long course and exposed position, are particularly susceptible to injury through compression against the cranial base. Collier is not thoroughly satisfied with this view, and submits, as a more likely explanation, the fact that the abducentes represent the only cerebral nerves which pursue a longitudinal rather than an oblique or transverse course from their superficial origin in the brain-stem to their dural foramina of exit. On this basis the shifting backward of the brain-stem—indicated by the well-recognized moulding of the medulla and cerebellar margin in the foramen magnum in cases of tumour (foraminal herniation)—would exert greater traction upon those nerves directed antero-posteriorly than upon those having a transverse course. To quote his exact words:—

> If the paralysis of these nerves is the result of shifting backwards of the brain-stem from supratentorial pressure, the effect being traction upon the nerves attached to the brain-stem, in proportion as their direction is more nearly a frontal-caudal direction, then the shifting backwards of the brain-stem would cause paralysis of the sixth nerve first, then of the third nerve, and lastly of the seventh and eighth, while the nerves which are transversely directed, or with sinuous course, would be little affected. Such traction would obviously be first manifested upon the side where supratentorial pressure is greatest—that is, upon the side of the lesion.

Even this ingenious hypothesis of Collier's does not give entire satisfaction, for, granting the downward dislocation of the brain-stem which is inevitable with any considerable increase of supratentorial pressure, it is nevertheless difficult to see, in view of the near proximity of their points of external origin, how this traction from dislocation could paralyse the abducens on one side alone without an extreme degree of lateral deformation of the pons. It is difficult, too, to reconcile this explanation of a palsy produced by the gradual traction to which the nerves would be subjected under these circumstances with the well-recognized fact that motor-nerves may undergo an astonishing degree of

stretching and elongation without any observable interruption of function. This is particularly well seen in cases of cerebello-pontine tumour in which there may not be the slightest evidence of weakness of the facial muscles unless the condition is far advanced, and yet post-mortem the seventh nerve may be found wrapped around the circumference of the tumour, flattened and drawn out to more than twice its normal length. The same thing may be seen in the case of the third and sixth nerves, which, much elongated and flattened, may similarly encircle a slowly enlarging infundibular tumour without any profound disturbance of ocular movements.

Peripheral motor-nerves therefore do not seem readily to lose their power of transmitting impulses when subjected to a slow process of elongation, such as would be occasioned by even the direct action of a new growth. We shall learn, too, from the study of our own cases that, in the absence of any direct affection of the nerves themselves or of their centres, a subtentorial (cerebellar) lesion which actually pushes the pons forward, with an effect consequently on the sixth pair the reverse of stretching, may nevertheless lead with even greater likelihood to a palsy of these nerves.

Character and Source of Material.

For the past five years my assistants and myself have been given the privilege by the pathological department of the hospital of making our own first-hand studies of the central nervous system of all patients who succumbed to the effects of intracranial lesions while resident under the surgical service. In addition to these opportunities we have had the good fortune to secure almost without exception the brains of all patients who have died elsewhere than in the hospital at varying periods after palliative tumour operations.

We have thus in the end come into possession of the brains of some forty of our 130 cases of operations for presumed brain tumour; and, in addition to this material, the tissues from a nearly equal number of cases which have died in the hospital from miscellaneous intracranial lesions—vascular disease, hydrocephalus, trauma, and the like—have been available for control of the matters under consideration.

It has been our invariable custom thoroughly to harden all brains *in situ* by carotid injections of formalin, so that the normal configuration of the parts should be absolutely preserved. Considerable deformation is an inevitable consequence of removing brains under tension while in

their original soft condition, and the additional custom of allowing the tissues to harden while suspended by the vertebral arteries would certainly destroy the neuro-vascular relationship to which we especially desire to call attention. Our main object in choosing this method of fixation has been to study the topographical relations of the intracranial lesions in the hope of throwing some light on their gross mechanical effects, rather than to investigate their histological characteristics or resultant degenerations—studies for which we are not particularly well trained.

We have endeavoured in each case to correlate the post-mortem findings with the ante-mortem symptomatology: first, by determining what localizing manifestations might have been expected from the given lesion and comparing them with the positive clinical findings; and, second, by attempting to determine the cause for such obscure or "distant" symptoms as had been recorded in the anamnesis or made out in the examination.

Naturally in this fairly large series of cerebral lesions there were a number of advanced cases in which so-called false localizing signs on the part of the ocular nerves had been observed, but, inasmuch as many of the tumours proved to be subtentorial, we were at first led to believe that the recorded periods of diplopia or the actually observed strabismus were due to a direct affection of the nuclei or to a possible stretching of the nerves, though neither of these interpretations explained the symptoms in all cases to our entire satisfaction.

Primary Observation.

Our suspicions that *arterial constriction* might possibly account for these "distant" symptoms in certain cases were first aroused in 1909 by the examination of the brain of a patient who for years had suffered from typical, though long unrecognized, uncinate gyrus attacks with fluctuating periods of left hemianæsthesia and hemianopsia (supposed to be hysterical), and, during the last year of her life, from recurring diplopia due to periods of weakness of the right external rectus. The brain showed (fig. 1) that a soft gelatinous growth extending from the uncinate region into the lateral recess had pushed the right abducens backward in such a way that it had become caught by and kinked around the right *A. auditiva interna*. When the encircling artery was lifted from its bed it showed not only a transverse constriction of the nerve, but also a deep grooving of the subjacent pons, much as though

an elastic band had been stretched around the brain-stem. Interesting, too, was the fact that the right oculomotorius (fig. 1), which was completely surrounded by the soft growth and much flattened, had never shown any clinical evidences of weakness, so that the conjecture was natural that the abducens palsy was more probably attributable to the mechanical constriction than to any possible compression from the growth.

This finding led to a review of the other brain-stems at our disposal, and although in some of them the vessels had been stripped away after the first gross external examination, or the tissues cut in such fashion as

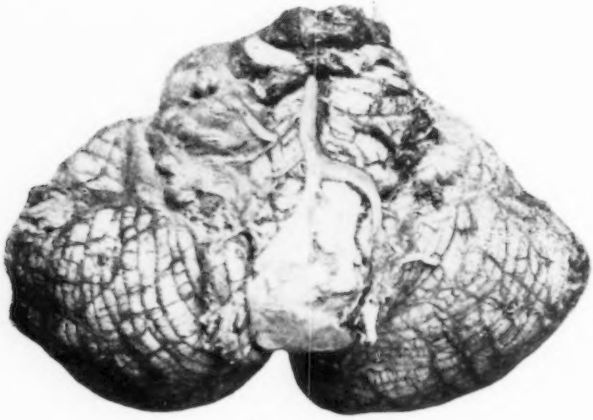

FIG. 1.

No. 10, Table I.—Brain-stem from first case observed. A gelatinous (degenerated) tumour of thirteen years' duration arising from the temporal lobe and spreading backward into the right lateral recess. Uncinate gyrus symptoms with right hemianopsia, right hemianæsthesia and right abducens palsy, all fluctuating in degree.

Note right third nerve flattened in tumour, but with no palsy, whereas the sixth is caught and constricted by right *A. auditiva int.*, over which it is buckled by pressure of tumour. Left *A. auditiva int.* has deeply grooved pons, but the nerve lay superficial to it. *Aa. cerebelli inf. ant.* both cross the pons caudal to the sixth pair.

to prevent a satisfactory examination for this particular neuro-vascular relationship, nevertheless in a large number of them the essential structures were preserved, showing in the majority a constriction often bilateral and at the same time similar to or even more marked than that observed in this first case. All of the brains acquired during the past year have naturally been investigated with this particular point in view.

Such a vascular strangulation of the abducens, needless to say, cannot occur unless the transverse arteries cross the pons superficial to the nerves, and, as will be seen, this is not the relationship which is usually given. As a matter of fact, the subject does not seem to have been given any especial consideration by anatomists, nor is there any reason why it should have been.

Anatomical Notes.

The fibres of the sixth pair emerge from the rhombencephalon, cephalad to the junction of the medulla oblongata and the pons, in a flat bundle consisting of from fifteen to eighteen small separate filia radicularia, which, as a rule, soon unite into the more or less rounded or flattened bundle characterizing the nerve in its longitudinal intracranial course.

The branches of the basilar artery (the only vessels which we need consider in relation to the sixth pair) pass transversely across the pons in a direction more or less at right angles with that taken by the abducentes. The two branches which come into direct relation with the nerve are: (1) The *A. cerebelli inferior anterior*, which passes outward and backward across the crus cerebelli, to be distributed to the anterior border of the under surface of the cerebellum, anastomosing with the posterior inferior cerebellar branch of the vertebral; and (2) the *A. auditiva interna*, which may either arise as a separate branch from the main basilar trunk or else through the bifurcation of the *A. cerebelli inferior anterior*. An average relationship is well shown in fig. 2.

Of the anatomies which I have consulted:—

Gray [4] does not mention the relationship of the inferior cerebellar artery to the nerve, but pictures (fig. 401) the nerve as superficial.

Quain [11] likewise does not mention the relationship, but also pictures (fig. 346, vol. ii, part 2, p. 412) the nerve as superficial.

Spalteholz [16] also makes no mention of the relationship, but pictures the nerve (p. 399) emerging between the *A. cerebelli inferior anterior* and the *A. auditiva interna* (an arrangement similar to our fig. 2, and one which would permit of strangulation of the nerve, by the larger artery, as in the conditions we are describing).

Henle [6] does not mention the relationship, but pictures (p. 125) the *A. auditiva* as superficial to the nerve, and the *A. cerebelli inferior anterior* as too far forward (i.e., arising near the trigeminal root) to bear any relation to the abducens. Practically this same picture occurs in Merkel's "Handbuch der Topog. Anat." (Braunschweig, 1885-90, vol. i, p. 143, fig. 83), and in Merkel's edition of Henle (4te Auf., 1901, p. 433 of Atlas).

Sappy [15] does not mention the relationship, but pictures (fig. 372) the arteries as superficial to the nerves at their points of emergence—a situation, in other words, at which they could easily escape from the effects of constriction.

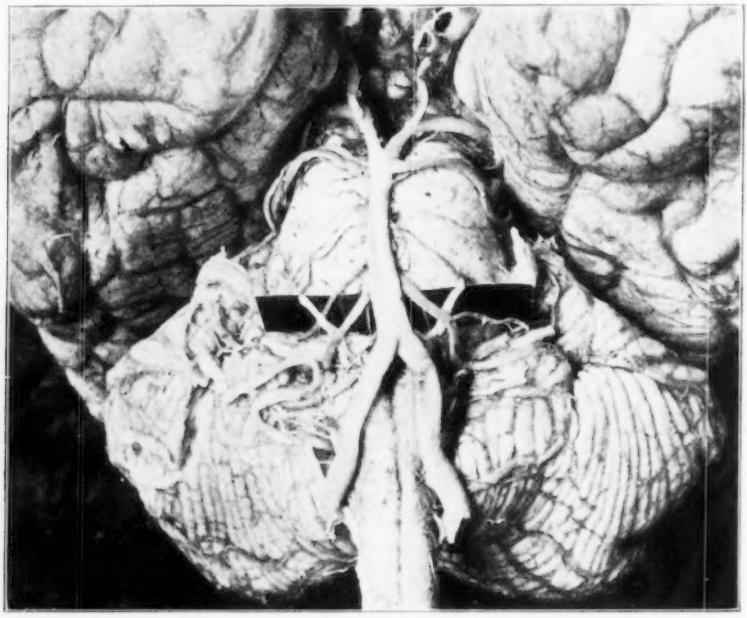

FIG. 2.

No. 36, Table II.—Brain-stem from a case of "pseudo-tumour" cerebelli (arteriosclerosis and nephritis causing cerebral œdema with typical pressure symptoms), showing a fairly normal distribution of the vessels, with minor anomalies (strip of black paper inserted under structures concerned).

Note: (1) Left *A. cerebelli inf. ant.* is superficial to but has not grooved the nerve, and after crossing it gives off the left *A. auditiva int.* A small (undescribed) minute arterial twig passes tailward from the *A. cerebelli inf. ant.* and has left a slight oblique groove in the left abducens.

(2) Right *A. cerebelli inf. ant.* is also superficial, and does not groove the right abducens; the anomalous twig is also present here, but does not overlie the nerve. The right *A. auditiva int.* arises as a separate vessel, and the nerve emerging between the two arteries lies superficial to it.

(3) The *A. cerebelli sup.* and the *A. cerebri post.* are widely separated. (The oculomotor nerves do not show in the photograph.)

Toldt [18] pictures (pp. 604-5) the nerves as crossed by both arteries in what proves to be their usual normal relationship. The matter is not mentioned in the context.

Rauber [12] does not mention the relationship, and pictures (pp. 794-5) the artery as superficial but crossing the nerves near their points of superficial emergence, in correspondence with Sappy.

Poirier and Charpy [10] are the only authors of works which I have consulted who make any special comment. They say that the nerve, several millimetres from its emergence, crosses the "middle cerebellar artery," passing sometimes below and sometimes above the vessel. These variations have been

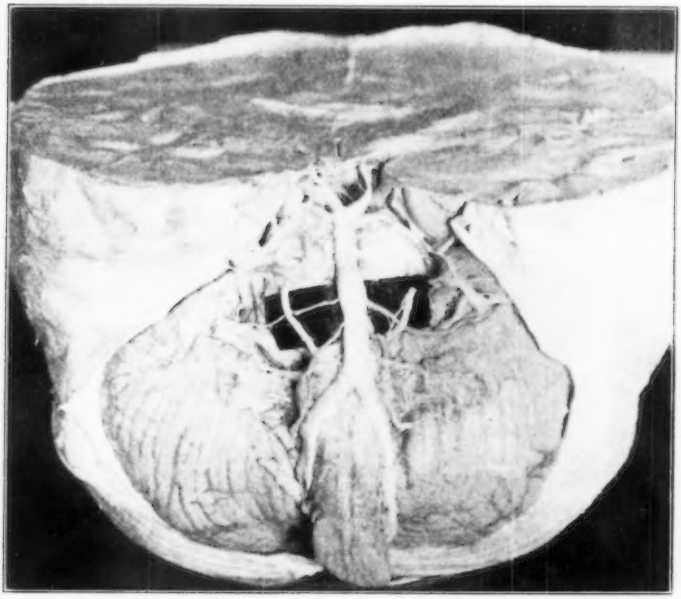

FIG. 3.

No. 7, Table I.—Gumma of pituitary body. No history of diplopia or evidence of ocular weakness on examination. Brain-stem shows no grooving of pons (black paper inserted to secure definition).

Photograph to show anatomical disposition of arteries. The *A. cerebelli inf. ant.* cross the abducentes near their points of origin and could hardly compress them. The right *A. auditiva* underlies the nerve; the left pierces it and gives off a small arteriole in passing. With such a disposition of the vessels, therefore "strangulation palsy" could not have occurred.

noted by Valentine, who has described an anomaly in which the artery passed through the separate bundles of fibres at the point of emergence of the nerves (*cf.* our fig. 3). In their description of the cerebral vessels, however (vol. iii, p. 563), the nerve is both described and pictured as being superficial to the artery.

It can be seen, therefore, that irregularities in distribution may occur, and that, owing to the presumed unimportance of the relationship, it has not been made an especial object of study. Contrary to the conditions pictured in most of the works cited above, one or the other of the arteries lies superficial to the nerves in the large majority of cases, and crosses them a short distance cephalad to their points of emergence.

Looking at the matter purely from an anatomical point of view, in the fifty-six brains that we have examined, thirty of which have been cases of brain tumour, the nerves on both sides have been almost without exception overlain by arteries in such a way that swelling or enlargement of the brain-stem, which would tend to stretch the transversely-disposed arteries, might be expected to produce a constriction of the nerve. Occasionally the arteries have branched off from the basilar so far posterior (as in the diagrams of Sappy and Rauber) as to cross the sixth pair at the level of, or caudad to, their points of emergence (*cf.* fig. 3). Even in these cases, however, the internal auditory arteries, if given off separately, may cross the nerves in such a way as to make strangulation possible. Not uncommonly, as in fig. 2 (right), the nerve may emerge, as pictured by Spalteholz, between the two arteries. In some cases one of the nerves has been found superficial to both arteries, but it is unusual for both nerves to be so disposed. In three cases out of the fifty-nine in our series, the artery has pierced the nerve (as in the anomaly described by Valentine, according to Poirier); in all three instances the anomaly occurred upon the left side (fig. 3).

Abducens Palsies Correlated with Possible Arterial Constriction.

In the following two tables have been gathered for reference the chief data relating to the neuro-vascular relationship presented by the fifty-nine brains which have been investigated with this point in view.[1] *Table I* includes thirty-nine observations—thirty-three cases of tumour and six of "pseudo-tumour" (four chronic abscesses) in which the

[1] In these tables, under the heading "Oculomotor Condition" (column 3), is recorded the presence or absence of subjective diplopia during the course of the disease, as elicited by the history, or as present at the time of examination; and also the presence or absence of any observable lack of parallelism of the globes from loss of muscle balance. In the absence of these the condition is noted as "normal."

Under "Anatomical Relationship" (column 5) is recorded the relative position of the arteries and nerves and the presence or absence of grooving. Thus, in *Case I* the *A. cerebelli anterior inferior* crossed the nerve on each side, but there was no grooving of the pons or of the sixth pair by the vessels. As no disturbance of the ocular muscle balance had been observed, the note in column 3 is "normal."

TABLE I.

Case No.	Surgical No.	Oculomotor Condition	Lesion	Anatomical Relationship
1	18,512	Normal	Large right cerebral glioma	Arteries superficial to nerves, but no grooving.
2	18,602	Normal	Small right parietal glioma	Arteries superficial to nerves, but no grooving.
3	18,640	No history of diplopia, but examination shows weakness of right sixth and third	Right cerebello-pontine endothelioma	Arteries removed from specimen: deep pontile grooving, especially on right, which does not affect nerves. Right third compressed between arteries.
4	19,854	Normal (?). (Patient unconscious)	Left frontal glioma	*Right:* artery superficial. *Left:* nerve superficial. Very asymmetrical arrangement; slight grooving.
5	20,286	Normal	Small cerebral tuberculoma; meningitis	Arteries superficial to nerves, but no grooving.
6	20,638	No history of diplopia. (Patient blind and unconscious.) Possible divergent squint	Enormous 400 grm. gliopsammoma, right hemisphere. Hydrocephalus. Diastasis of sutures	Arteries superficial: practically no grooving. Third pair compressed between arteries in much distorted position.
7	21,026	Normal	Pituitary gumma. Arterio-sclerosis	*Right:* nerve superficial. *Left:* artery pierces nerve. No grooving (fig. 3).
8	21,400	Normal	Cerebellar cyst	*Right:* nerve superficial. *Left:* artery superficial. Considerable grooving of pons, escaping sixth pair. Third pair slightly indented by arteries.
9	21,411	History of diplopia	Multiple cerebral melanotic sarcomata	Arteries superficial: right constricts nerve; left caudad to nerve. Moderate grooving
10	21,654	History of periodic diplopia	Gelatinous glioma (?) of uncinate region and lateral recess (right)	Arteries superficial. Deep grooving, especially on right (fig. 1).
11	21,915	No recorded diplopia. (Patient unconscious.) Obvious squint	Cerebellar cyst	*Right:* artery superficial. *Left:* nerve superficial. Deep grooving, which escapes nerves. Third pair kinked and grooved by arteries (fig. 4).
12	22,120	No history of diplopia. Presumable but unrecorded involvement of third and sixth pairs before death	Glioma brain-stem	Arteries superficial: deep grooving, from which abducentes largely escape. Third pair implicated. Nuclear involvement possible (?).
13	22,428	History of diplopia. Convergent squint	Cerebellar tubercle	*Right:* arteries superficial and cut deeply. *Left:* abducens emerges cephaled to *A. cerebelli inf. ant.*, but is overlain and cut by *A. auditiva*. Pons and nerves deeply grooved (fig. 9-13).
14	22,488	Normal	Left temporal endothelioma	Arteries superficial, though asymmetrically disposed. Practically no grooving of pons or nerves.

TABLE I. (*continued.*)

Case No.	Surgical No.	Oculomotor Condition	Lesion	Anatomical Relationship
15	22,551	A transient ptosis with temporary palsy of left third nerve during epileptic attacks	Glioma left hemisphere. Internal hydrocephalus	Arteries superficial, crossing nerves at their points of emergence. Practically no grooving.
16	22,691	Diplopia: left internal squint	Tumour (glioma) compressing side of medulla near foramen. (No increase of tension)	*Right*: abducens involved in tumour. *Left*: artery caudad and superficial; no grooving.
17	22,812	History of recurring diplopia. Bilateral internal strabismus	Right frontal glioma	Arteries symmetrical and superficial to nerves. Deep constriction of left abducens: slight, of right. Third pair compressed between arteries.
18	22,867	Normal	Glioma of temporal lobe	*Right*: nerve superficial. *Left*: *A. auditiva* superficial, with slight grooving
19	22,896	Normal	Tempero-sphenoidal glioma	Arteries superficial. Slight grooving.
20	23,014	History of diplopia. Convergent strabismus	Glioma of quadrigeminal region	Arteries superficial, with grooving. Possible nuclear involvement (?).
21	22,950	Normal	Glioma temporal lobe and uncinate gyrus	(Right relationship lost in sections.) Left artery perforates nerve. Grooving inconspicuous.
22	23,967	Normal	"Pseudo-tumour." Chronic abscess occipital lobe with pressure symptoms	*Right*: artery superficial. *Left*: artery perforates nerve. No grooving
23	23,850	Normal	Glioma left hemisphere. Extensive œdema	Arteries superficial with practically no grooving. Very asymmetrical arrangement.
24	24,067	Normal	"Pseudo-tumour." Old abscess left hemisphere	Arteries superficial and symmetrical. No grooving.
25	24,137	No history of diplopia recorded	Uncinate glioma	Arteries superficial and asymmetrical. Considerable grooving: left branches by common trunk crossing near point of emergence.
26	24,307	Normal	"Pseudo-tumour." Old abscess frontal lobe	Arteries superficial, with typical arrangement. No grooving.
27	23,967	Normal	"Pseudo-tumour." Chronic abscess left occipital lobe	*Right*: artery superficial. *Left*: nerve superficial. Considerable grooving.
28	24,357	Normal	"Pseudo-tumour." Hydrocephalus	Nerves superficial. No grooving.
29	24,398	Diplopia. Convergent squint	Cerebellar "neurocytoma" (Mallory)	Arteries superficial and symmetrical. Marked grooving (figs. 6, 7, 8).
30	24,413	History of diplopia	Glioma corpus callosum. Extensive œdema	Arteries superficial; symmetrical, and deeply groove nerves and pons.
31	24,673	Diplopia with divergentsquint. (Third nuclear involvement?)	Metastatic carcinoma of cerebrum	Arteries superficial to nerves, but no grooving.

TABLE I. (*continued.*)

Case No.	Surgical No.	Oculomotor Condition	Lesion	Anatomical Relationship
32	24,714	Normal. Some weakness of conjugate movements to left	Cerebellar glioma ..	*Right:* artery superficial to nerve. *Left:* abducens emerges between *A. cerebelli inferior* and *A. auditiva*. No grooving.
33	25,224	Right abducens palsy with conjugate paralysis	Cystic glioma left lateral recess	*Left:* artery superficial and deeply grooves nerve and pons. *Right:* nerve in tumour (fig. 5).
34	St. Agnes	No history of diplopia. (Homonymous hemianopsia).	Glioma left parietal lobe	Arteries superficial and slightly groove nerves.
35	24,886	Normal	Multiple small metastatic carcinomata; cerebral and cerebellar. (No increase of tension.)	*Right:* artery superficial. *Left:* nerve superficial. Practically no constriction.
36	23,479	Normal	"Pseudo-tumour." Epilepsy	Arteries superficial but no grooving.
37	Church Home	Diplopia. (Nuclear involvement.) Blindness. Divergent squint	Glioma quadrigeminal region	Arteries superficial but no grooving (fig. 2).
38	23,953	History of diplopia. Strabismus variable. (Patient unconscious.)	Cerebellar cyst. Internal hydrocephalus	Arteries superficial. Marked grooving.
39	26,128	No diplopia.. ..	Frontal endothelioma. Extreme pressure	Considerable grooving of pons, especially by vertebrals. Abducentes escape, as nerves emerge caudad to crossing.

TABLE II.

Case No.	Surgical No.	Oculomotor Condition	Lesion	Anatomical Relationship.
40	17,468	Normal	Fracture skull. Intracranial hæmorrhage	Arteries superficial to nerves and symmetrical. No grooving.
41	18,369	Normal. Right conjugate deviation	Cerebral contusion with extravasation over frontal lobes	Arteries superficial and symmetrical. No grooving.
42	18,956	Normal	Internal hydrocephalus. (Infant)	*Right:* artery superficial. *Left:* nerve superficial. No grooving.
43	19,644	Not observed ..	Suppurative meningitis with obstructive hydrocephalus	Arteries superficial and symmetrical. No grooving.
44	20,486	Patient unconscious	Capsular apoplexy ..	Arteries superficial and very sclerotic, but no grooving.
45	21,493	Patient unconscious	Capsular apoplexy ..	Arteries superficial; extensively diseased. No grooving.

TABLE II. (*continued.*)

Case No.	Surgical No.	Oculomotor Condition	Lesion	Anatomical Relationship
46	21,844	Patient unconscious	Tuberculous meningitis. Hydrocephalus	Arteries cross at pontomedullary junction and escape nerves. No grooving.
47	21,942	Normal	Meningitis (Wichselbaum). Hydrocephalus	Arteries superficial but asymmetrical. No grooving.
48	21,980	Normal (?)	Capsular apoplexy ..	Arteries superficial; suggestion of grooving.
49	23,278	Patient unconscious. Conjugate deviation to right	Fracture of base. Intracranial hæmorrhage	Arteries superficial and symmetrical. No grooving.
50	23,370	(Newborn)	Meningitis after forceps delivery with fracture	Arteries superficial and symmetrical. No grooving.
51	23,690	No abnormality observed. Patient unconscious	Apoplexy. Extensive basilar sclerosis	Sclerotic arteries superficial. No grooving of pons or nerves.
52	24,379	No abnormality observed. Patient unconscious	Epilepsy: chronic arachnoiditis	Sclerotic arteries superficial and symmetrical; cross nerves 5 mm. from origin. No grooving.
53	24,203	No abnormality observed	Tuberculous meningitis	Arteries superficial. No grooving.
54	24,290	Normal (?). Conjugate deviation	Encephalitis chronica. Epilepsy	Arteries superficial, but no grooving of nerves or pons.
55	23,398	Patient unconscious. Right conjugate deviation, head and eyes	Encephalitis acuta. Septicæmia	*Right:* artery superficial. (*Left:* relation destroyed.) No grooving.
56	24,307	Normal	Abscess frontal lobe: chronic	Arteries superficial: typical. No grooving.
57	Washington case	Unrecorded.. ..	Microgyria: adiposis dolorosa	Arteries superficial: typical. Suggestion of grooving.
58	Church Home	Unrecorded.. ..	Chronic hydrocephalus	Arteries superficial, with irregular distribution. No grooving.
59	24,337	Normal	Cerebral œdema ..	*Right:* nerve superficial. *Left:* artery superficial. No grooving.

pressure phenomena were identical with those characterizing an actual neoplasm. *Table II* comprises twenty observations on brains with miscellaneous lesions which clinically could not have been mistaken for tumour. Most of them represent acute conditions in which there was no history of diplopia or observable squint, so that they are valuable largely from the standpoint of anatomical relationship, though they show at the same time the rarity of arterial grooving in brains unaffected by the long enduring pressure effects of tumour.

ANALYSIS OF TABLES.

These tables are of interest from three points of view: one concerns the topographical relation of the arteries and nerves; another, the

presence or absence of grooving produced by the vessels; the third, the association of recorded diplopia or squint with the presence or absence of grooving.

(1) *Anatomical Relationship.*

It will be seen that in the large majority of the fifty-nine cases the arteries are more or less symmetrically disposed, and overlie the abducentes in such a way as to render constriction possible. In a number of instances, as in Nos. 15, 39, and 46, the arteries cross so far posteriorly as to lie in the ponto-medullary groove (fig. 3, for example), or else so near the points of emergence of the sixth pair as to make it seem unlikely that the nerves could have been affected by the constriction. In other instances the disposition of the vessels was very asymmetrical, and yet one or the other of them, or the combined trunk, crossed the nerve a few millimetres cephalad to its point of emergence.

In no cases were both of the arteries found to underlie the nerves on the two sides, after the fashion usually depicted in the text-books. There were ten cases, however, in which the nerve on one side alone was superficial to the two arteries considered, or to their combined trunk in case they arose from a common stalk. Of these ten cases the right abducens was superficial in three instances (Nos. 8, 18, and 59), and the left in six (Nos. 4, 9, 11, 25, 27, 35, and 42). In three cases the left abducens was perforated—in Nos. 21 and 22 by the *A. cerebelli anterior inferior*, and in No. 7 by the *A. auditiva* (fig. 3), and thus only a single bundle of its fibres could have been constricted. Occasionally, as in Nos. 13 and 32, it was the *A. auditiva* which, on one side at least, overlay the nerve; whereas the *A. cerebelli anterior inferior* crossed the pons caudad to the point of its emergence.[1]

(2) *Arterial "Grooving."*

The most striking feature of the brain-stems of the tumour cases, when once attention has been called to it, is the deep grooving, particularly of the pons, which is produced not only by the branches in which we are chiefly interested, but also by the basilar and its *rami ad pontem*

[1] From the anatomist's standpoint it would be desirable to examine a larger series of brains than these pathological ones which I have had at my disposal, and particularly injected brains, which would render the investigation of the relationship—provided the tissues were removed with careful preservation of the sixth pair, which are often torn out—much easier than in the case of the bloodless (through perfusion) vessels in my specimens. Aside from the mere demonstration that the topography as usually given is erroneous, the chief value of my tumour material lies in the evidences furnished by these tissues of the deep grooving.

(fig. 4). The transverse vessels may have cut so deeply into the tissues as actually to lie below the level of the surface. Though difficult to photograph, the grooving is fairly well represented by the shadows,

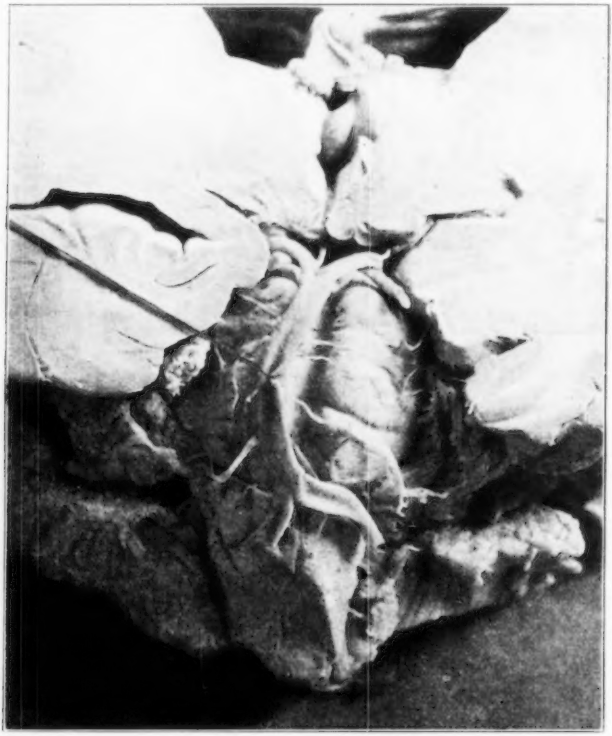

FIG. 4.

No. 11, Table I.—Cerebellar cyst with secondary internal hydrocephalus. Death following lumbar puncture. Patient had a squint affecting third pair and right abducens.

Photograph shows brain-stem with basilar artery lifted from its deep longitudinal groove. All lateral branches also have cut deeply into pons.

Note: (1) Right *A. cerebelli inf. ant.* is superficial to and grooves the abducens.

(2) On the left the abducens is superficial to this artery, which, however, has deeply cut the pons; but it is overlain by the left *A. auditiva int.*, the deep groove made by which can be seen leaving the basilar groove on the right near the medulla.

(It is to be noted that the vessels have been washed free of blood by the fixative agent, and hence are difficult to photograph clearly.)

particularly in figs. 4 and 9. It is to be noted that these arteries are not stiffened and sclerosed, but are normal vessels.

In the fifty-nine brains, definite grooving was present in twenty-six cases, twenty-five of these being cases of tumour. Inasmuch as the method of hardening and removal of the brains was the same not only for the tumour cases but for the miscellaneous group as well, it is improbable that the vessels could have become sunken into the tissues purely as a result of the method of preparation.

Furthermore, of the eighteen out of the thirty-nine tumour cases which showed no grooving, nearly all were cerebral lesions, and all but one (No. 27) of the "pseudo-tumours" (abscess, hydrocephalus, epilepsy, &c.) fall likewise within this group. Of the twenty-one tumour cases in which grooving occurred, it was pronounced in fifteen, ten of which were subtentorial lesions. Deep grooving occurred, therefore, in only five supratentorial cases (Nos. 17, 25, 27, 30, and 39). Pressure was extreme in all of these cases, and in two of them—cases with excessive vomiting—the cerebellum showed post-mortem obvious signs of œdema. In seven of the twenty-one tumour cases showing grooving it was comparatively slight in degree (Nos. 4, 9, 18, 19, 20, 21, and 34), and in all but one of these cases (No. 20, a glioma of the quadrigeminal region) the lesion was cerebral and more or less remote from the brain-stem. The same is true of the remaining eighteen of the thirty-nine tumour cases in which no grooving was present, the lesions, with only three exceptions, being cerebral and remote. The three exceptions are notable, for one of them (No. 16) was a fibrous tumour compressing the side of the medulla, the patient having never shown any general pressure symptoms other than excessive vomiting; another (No. 35) also showed cerebellar involvement, but there was practically no increase of pressure and no clinically recognizable cerebellar symptoms had been detected; and the third (No. 37) was a glioma of the quadrigeminal region in which pressure symptoms had likewise been inconspicuous—a rapidly progressing lesion with pronounced ataxia and bilateral nuclear blindness and deafness.

In three of the tumour cases (Nos. 12, 20, and 37) there was more or less diffuse oculomotor paralysis, evidently from direct nuclear affection of the third pair at least; but inasmuch as there was no accompanying weakness of the facial muscles, and in view of the obvious grooving, it seems not unlikely that the paralysis of the abducentes in these cases was peripheral and due to vascular constriction rather than to a nuclear involvement. On this view, a strangulation palsy of the sixth pair might be superimposed on a nuclear involvement of the third pair, thus accounting for some of the complex ocular palsies in pontine tumours which affect the *Nn. oculomotorii* and the *Nn. abducentes* with-

out clinical evidence of impairment of the neighbouring nuclei.[1] An illustration of a possible abducens palsy due to constriction on the opposite side to the lesion is shown in fig. 5.

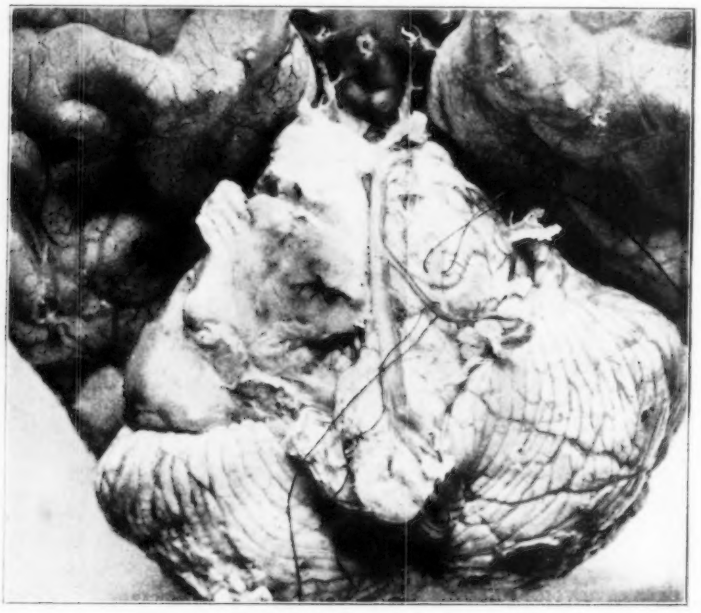

FIG. 5.

No. 33, Table I.—Infiltrating cystic glioma of right lateral recess and pons, with affection of many of the right cerebral nerves (third to tenth) and of the left abducens. Note deep serpentine groove of left side of pons crossing the nerve, made by the artery which has been drawn aside by thread.

[1] Uhthoff [19] calls attention to the frequent affection of the abducens in cases of pontine tumour, stating that only in 18 per cent. do the nerves escape. It is relatively rare for the abducens to remain the only nerve affected during the further progress of the disease, but it is usually the earliest one to be involved; and in about half of his cases the lesion was bilateral. In the early stages of a pontine tumour it can long remain an entirely isolated manifestation of the trouble. As the lesion advances, the facial, owing to its nuclear proximity, becomes affected, in 75 per cent. of the cases, but in one-third of these the abducens palsy is bilateral, whereas the facial involvement remains unilateral. It is notable that the trochlear is very rarely involved in these lesions—a nerve which is in such a situation that it cannot easily be implicated, at least by arterial pressure. In the case of tumours in the neighbourhood of the fourth ventricle, Uhthoff notes that associated paralyses of the ocular nerves are very much less common—only four times in sixty-two cases. The abducens, however, is affected in 25 per cent. of lateral recess tumours—more frequently than any other nerve. In more than half of the observed cases the paralysis was bilateral, which would speak in favour of the arterial constriction theory. The third and fourth pair are scarcely ever affected.

Briefly, one may say that the arterial grooving which we are considering is relatively rare in brains not the seat of true tumour, and that when it occurs it is more common in the presence of subtentorial lesions than in those situated in the cerebral hemispheres.

(3) *Relation of Diplopia to Grooving.*

Now, looking at these tables from the standpoint of recorded diplopia or observed squint, it will be seen: (1) That an existent ocular disturbance could not be accounted for on the basis of arterial constriction in only three cases, two of them cerebral (Nos. 31 and 37), both cases of presumed third nuclear involvement and divergent squint, and one a cerebellar case (No. 16), in which there was no grooving; and the recorded left internal squint remains unaccounted for on any basis. (2) That the ocular disturbance could be best accounted for by arterial constriction in eleven cases, of which nine were cerebellar and two cerebral; and (3) that an absence of ocular disturbance in the presence of grooving usually indicates either a caudad position of the constricting vessels or else superficially disposed nerves.

Conspicuous grooving such as might well explain an existent convergent squint was present more often in the cerebellar than in the cerebral cases; and the instances of deep grooving, it may be added, were most often associated with a fixed strabismus, those of slight grooving merely with a history of occasional diplopia.

It is to be noted, furthermore, that only four out of the fourteen subtentorial cases were free from grooving (Nos. 16, 32, 35, and 37). Nos. 16 and 35 were notably free from manifestations of increased tension; No. 37 was a rapidly growing glioma of the quadrigeminal region, which led early to incapacitating paralyses; No. 32 therefore remains as the only striking exception. On the other hand, fourteen out of the twenty-five cerebral (supratentorial) cases showed no grooving whatsoever—an indication of the relative freedom from such swelling of the hind-brain as is necessary under these circumstances to produce the form of vascular strangulation under discussion. A certain measure of grooving, it must be added, was present in seven of the cerebral (supratentorial) cases (Nos. 4, 18, 19, 21, 25, 34, and 39) with no clinical record of diplopia. In only two of these (Nos. 25 and 39) was the vascular grooving at all pronounced, and in both cases the abducentes presumably escaped from constriction owing to a caudal position of the vessels. Nevertheless, they may both be regarded as possible exceptions to the rule, for the two factors making diplopia possible

through vascular strangulation are present—namely, deep grooving and superficially disposed arteries. In the other five brains the grooving is possibly too slight to have affected the nerves seriously, and Nos. 4, 18, 21, and 34 are explicable on other grounds.

It is natural, of course, that a certain degree of grooving by superficially disposed vessels may not actually suffice to produce weakness of the external rectus at any time in the course of the disease, for doubtless the constriction must be considerable to accomplish this result. When the strangulation is on the border-line of producing functional weakness

FIG. 6.

No. 29, Table I.—Patient, looking to left, showing double abducens palsy; typical cerebellar symptoms with hydrocephalus which had caused diastasis of sutures.

of the nerves, subjective diplopia, with or without clinically observable squint, may fluctuate in correspondence with varying degrees of tension. All who have a wide experience with these patients are familiar with cases in which periods of extreme headache, leading up to attacks of vomiting, are accompanied by double vision, which disappears

FIG. 7.

No. 29, Table I.—Grooving of both *Nn. abducentes* by symmetrically placed and superficially disposed *Aa. cerebelli inf. ant.*, which give off the *Aa. auditivæ*.

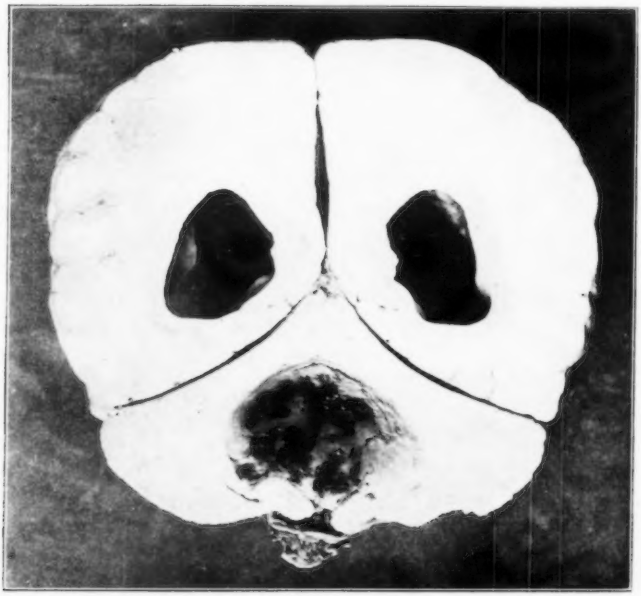

FIG. 8.

No. 29, Table I.—Coronal section posterior to medulla (looking forward) to show mesial tumour, a neurocytoma, not involving motor nuclei.

when the paroxysm subsides. These paroxysms are doubtless due to waves of increased pressure from vascular stasis or neighbourhood œdemas, or varying grades of obstructive hydrops ventriculorum. They are often brought on by some unusual effort, by a change of posture, or, indeed, may occur unaccountably.

One would scarcely expect to find that the clinical history records a bilateral palsy of equal degree, even though, post-mortem, both nerves show evidence of grooving, for the arteries are rarely disposed with such perfect symmetry as shown in fig. 7; and even should they be, the constriction on one side or the other might fall short of a total physiological "block" to the transmission of impulses. A functional interruption of this sort, as experimental and clinical observations have taught us, may not necessarily be accompanied by a demonstrable degeneration of nerve fibres. The well-recognized fluctuating type of diplopia, with a periodic convergent squint, may doubtless be attributable to temporary interruptions from this cause, and it is to be noted that normal parallelism of the globes has promptly returned in a number of cases after a successful decompression. Doubtless, when the constriction is extreme and of long standing (as in No. 13), permanent organic changes in the nerve will occur. The following cases may serve as examples to illustrate some of these matters in greater detail:—

Case No. 29 (Surgical No. 24,398).—*Median cerebellar tumour with double convergent squint. Symmetrically disposed vessels producing bilateral grooving of both abducentes. No degeneration.*

The patient, a child aged 9, admitted July 9, 1909; had fallen, producing a frontal scalp wound five years before admission. History otherwise unimportant.

Present symptoms observed for six months: frontal headaches, staggering gait, and rolling of head; recurrent projectile vomiting. Owing to static ataxia has been unable to stand or walk for the past three months. Child has been "cross-eyed" for several weeks (fig. 6).

Examination.—Apparent diastasis of cranial sutures. "Cracked pot" sound on percussion. (Internal hydrocephalus?) Dilatation of extracranial veins. Slight exophthalmos. Suboccipital tenderness.

Cranial Nerves.—Anosmia: 7 D. of choked disc with loss of visual acuity to $\frac{1}{60}$ and colour interlacing, though but slight constriction of fields: bilateral convergent squint: weakness of left seventh: impairment of right eighth, with tinnitus; otherwise negative.

Cerebral lobes.—Negative in all respects, except for slight mental inactivity and forgetfulness.

Hind-brain.—Pronounced ataxia of extremities, particularly on the left;

inability to assume and hold upright position standing or sitting. Nystagmus in all directions.

Diagnosis.—Intracerebellar tumour.

Operation.—July 13, 1909. Exposure of a median, well-encapsulated cerebellar tumour. Case difficult, owing to extreme tension and unsatisfactory primary evacuation of cerebrospinal fluid. On account of the extensive protrusion an attempt to enucleate was forced on the operator at this stage. Removal only partially accomplished before medullary symptoms set in. Closure of wound. Death three hours later, with hyperpyrexia.

Autopsy.—Brain hardened *in situ* as usual. Grooving of abducentes (fig. 7). The tumour (pronounced a neurocytoma by Dr. F. B. Mallory) occupied a median position in cerebellum (fig. 8) extending upward towards the vermis. Nuclear region in floor of the fourth ventricle is free from gross lesion, but might have suffered from compression, though tumour lies posterior and remote.

Careful histological study of the sixth pair shows fairly deep grooving, but no degenerated fibres on either side of the constriction are demonstrable. Constricting vessels normal.

Case No. 13 (Surgical No. 22,428). *Multiple tuberculomata. Ocular palsies without direct nuclear involvement. Deep bilateral abducens strangulation by A. auditiva interna, with extensive degeneration.*

The patient, a man 29 years of age, admitted to Dr. Barker's service, May 9, 1908, with pleurisy and laryngitis, recognized for some months as tuberculous. Pyrexia and loss of weight.

Intracranial Symptoms.—Indefinite headache for the past month. At present, intense posterior headaches with vomiting and choked disc. Subjective numbness in right face and fingers. Weakness of the lower right face. Diplopia, with obvious lack of parallelism in globes and weakness of conjugate movements (pupils equal and reactions normal). Otherwise practically nothing. Complete absence of evidences of cerebellar lesion other than occipital headache and some resistance to movements of neck.

Diagnosis.—Intracranial tuberculosis: character uncertain.

A rapid advance in pressure symptoms, with prostrating headaches, made it seem advisable to attempt a decompression, though the seat and nature of the lesion remained obscure.

Operation.—May 22, 1909. Attempted subtemporal decompression. Anæsthetic badly taken; cyanosis. Extreme tension of dura. Though aware of the risk, attempt was made to diminish tension by lumbar puncture, a cerebellar lesion not being suspected. Fluid under great tension; sudden cessation of flow after 7 c.c. withdrawn. Immediate respiratory failure. Temporary resumption of spontaneous breathing after opening dura at established subtemporal bone defect. Secondary cessation. Death after several hours of artificial respiration.

Autopsy.—Brain hardened in the usual fashion. Extreme foraminal

protrusion of cerebellum and medulla (fig. 9). Basilar vessels normal, but deeply groove the pons. Constriction of both abducentes extreme; third pair also deeply indented by closely placed posterior cerebral and anterior cerebellar branches. On section brain shows multiple solitary tuberculomas; one fairly large growth (2 cm. in diameter) situated in left cerebellum; extensive cerebral and cerebellar œdema. Nuclear region in floor of the fourth ventricle free from gross lesion.

A histological examination of the nerves by Dr. Charles M. Byrnes shows an extensive degeneration of fibres on both sides of the constriction, at which point destruction and replacement by scar tissue has been extensive. The marked constrictions produced by the arteries is well shown in figs. 10 and 12, and the degenerations in figs. 11 and 13. The vessels are normal.

In the hope that some clinical relationship might be established between a recorded diplopia (particularly when due to a convergent squint) and the situation of the lesion, a cursory review has been made of the histories of eighty-one cases of brain tumour—cases in which the lesions have been either demonstrated or removed at operation, or, in the presence of an unlocalizable lesion, cases in which a simple decompression has been performed. Stress cannot be laid upon the exactness of the figures, for there are many possible sources of error; thus, though subjective diplopia may have been recorded in the anamnesis, the patient may not have understood the meaning of "double vision," or it may not have been inquired into; the "distant" symptom of oculomotor palsy is late in occurrence, and many patients were decompressed at an early stage; confusing oculomotor palsies, central or peripheral, may have been present in combination with an abducens weakness; the diplopia in some cases, as in the hypophysial lesions, was obviously not due to a strangulation palsy, and so on. Nevertheless, in some respects the figures are not uninteresting in connexion with the cases included in Table I, in which the arterio-vascular relationship was demonstrable after death.

Of the eighty-one cases, fifty-four represent cerebral, or presumably cerebral, and twenty-seven cerebellar, or presumably cerebellar, lesions. Nineteen, or 75 per cent., of the subtentorial (cerebellar and pontine) cases showed a positive squint on examination or gave a definite history of periods of diplopia. Eight, or only 15 per cent., of the cerebral cases gave such a history or showed squint, and in all but three of these the condition can be attributed to a direct peripheral involvement of the nerves in a basilar growth which had led to multiple palsies. One would naturally expect that the subtentorial lesions would be more likely to produce diplopia from direct nuclear involvement (as in the pontine tumours,

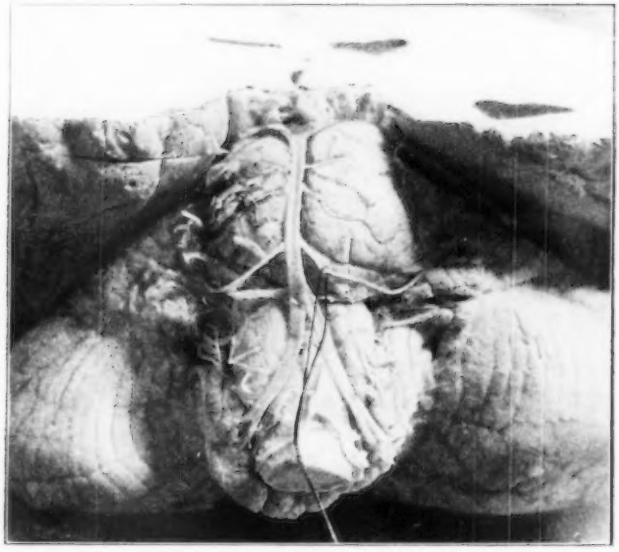

FIG. 9.

No. 13, Table I.—Tuberculoma of left cerebellar hemisphere and multiple tuberculomas of cerebrum with œdema and greatly increased tension. Fluctuating palsies of the sixth pair. Marked foraminal herniation.

Note: (1) Pons much grooved by all vessels, and even the abducens themselves have left imprints (*cf.* on right where nerve is turned out of bed).

(2) On the left the two arteries arise from a single trunk which is superficial to the abducens and has deeply cut into pons and across the nerve (artery held away by black thread).

(3) The right abducens is also crossed and constricted by both *A. auditiva int.* and *A. cerebelli inf. ant.*, which crosses the nerve just caudad to the ponto-medullary groove (*cf.* figs. 10-13).

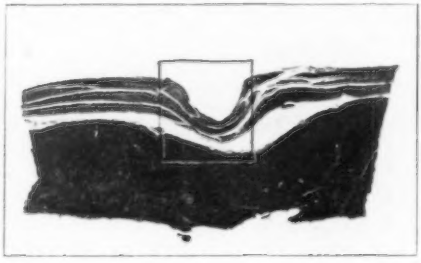

FIG. 10.

No. 13.—Longitudinal section of *N. abducens sinistra* showing imprint of *A. cerebelli ant. inf.* Squared area enlarged in fig. 11. Hæmatoxylin and eosin stain (*cf.* fig. 9).

FIG. 11.

No. 13.—Enlargement of area squared in fig. 10 to show cicatrization of bundle which lay in juxtaposition to artery and much degeneration of fibres of other bundles. Weigert stain; yellow areas screened out from negative.

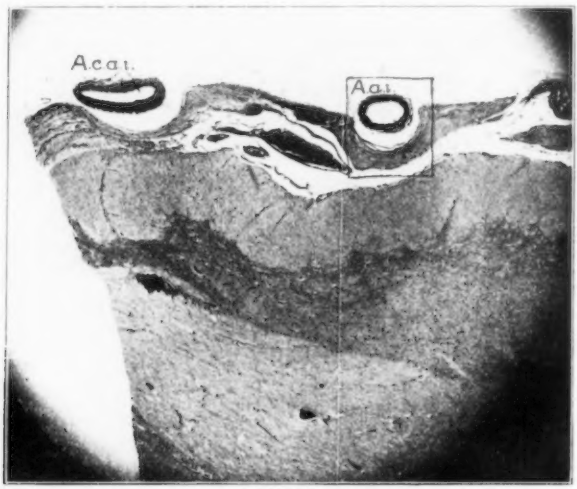

FIG. 12.

No. 13.—Longitudinal section of *N. abducens dextra* showing transverse indentation made by *A. cerebelli ant. inf.* (A.c.a.i.) and by *A. auditiva int.* (A.a.i.) (*cf.* fig. 9). Squared area represents enlarged field in fig. 12. Hæmatoxylin and eosin stain.

of which there were four instances), or from direct peripheral affection due to pressure of a cerebello-pontine endothelioma, of which there have been six examples. In nine of the nineteen cerebellar cases the squint disappeared after the operation, together with the subjective diplopia. Some of these cases represented simple suboccipital decompression; in others the growth was removed, and it is unlikely that this could have been accomplished without permanent damage to nerve or centres if they had previously suffered from an actual

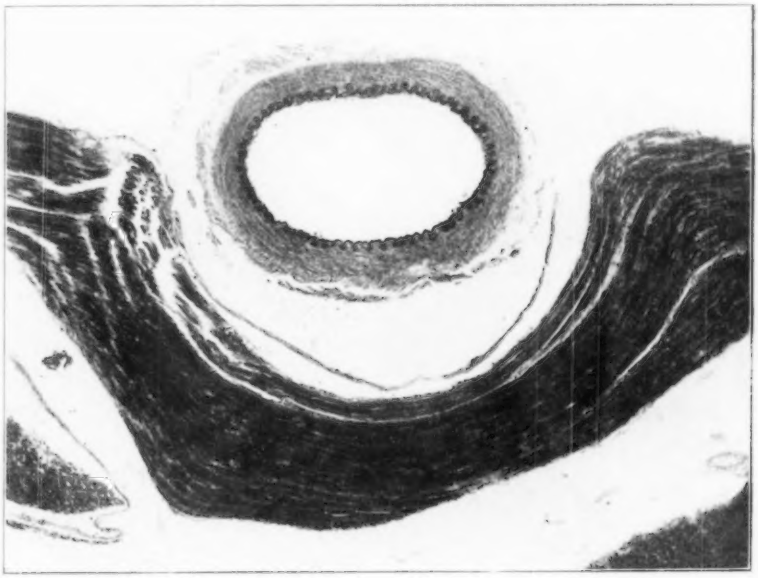

FIG. 13.

No. 13.—Enlargement of area squared in fig. 12 to show degenerated fibres and normal arterial wall. Weigert stain: yellow areas screened out in photograph.

"neighbourhood" lesion rather than from some "distant" or secondary process.

IN SUMMARY.

It seems fair to say, therefore, (1) that in the usual topographical relationship the arteries are superficial and cross the nerves in such a way as to render constriction possible; (2) that in the majority of

the fatal cases in which diplopia (particularly in association with a conjugate strabismus) had been recorded, the pons was grooved and the abducentes constricted by the arteries; (3) that these symptoms were much more common in the cerebellar cases, though not alone restricted to them; (4) that the cerebellar lesion, though often so remote as to render unlikely any possible direct affection of the nerves themselves or of their centres, nevertheless, owing presumably to swelling and enlargement of the hind-brain, produced such a stretching of the arteries around the brain-stem as to constrict and groove the nervous tissues, much as though a rubber band had been placed around them; (5) that in cases of extreme constriction the nerves may be found degenerated and replaced by scar-tissue at the point of constriction; and, finally, (6) that the "distant" symptom of a convergent squint may disappear after the successful removal of a cerebellar tumour whose situation was too remote to have directly affected the peripheral neurones of the sixth pair.

If, on this basis, abducens palsies may in some cases be attributable to arterial constriction, they may in a sense be regarded as one of the general manifestations of pressure or tension; a convergent squint, furthermore, like each of the other so-called general pressure symptoms, may have a certain localizing value as speaking in favour of a lesion of the hind-brain.

Abducens Palsies in Other States.

It is well known that in many conditions besides tumour the sixth pair exhibit a particular susceptibility to injury. This is the case in fracture of the cranial base, in otitis media, cerebral arterio-sclerosis, hydrocephalus, and the like.[1] Köllner [7] has recently made a study of 250 cases of abducens palsies occurring either alone or in combination with lesions of the other ocular nerves. There were 13 instances with tumor cerebri, 13 following trauma, 70 with tabes, and 40 with lues: the rest were due to more or less scattered lesions. Doubtless, in the large majority of the syphilitic cases, the nerve was directly involved in a gummatous meningitis. However, judging from the fact that in none of Köllner's series was the diagnosis confirmed by a subsequent section, it is not improbable that a number of the cases classified as lues were more properly tumours. This at least would conform with our experience with the clinical diagnosis of intracranial lues. In eight of the 13 supposed

[1] A number of the conditions apt to be associated with paralysis of the sixth pair are cited at length by Ricaldoni [13].

tumour cases the oculo-motorius was involved, in company with the abducens.

Uhthoff [19] has given some statistics of the ocular disturbances accompanying hydrocephalus and meningitis serosa. A convergent strabismus was by far the most common symptom occurring in 26 cases in his series, of which number nine came to autopsy. A direct nuclear affection was demonstrable in only one case, the paralyses in the others being regarded as a form of pressure palsy to which the abducens is susceptible "owing to its long intracranial course." A combination of abducens and other palsies was relatively rare in these cases. The author speaks of the occasional subsidence of the ocular disturbances after the withdrawal of the fluid by a lumbar puncture.

An unaccountable paralysis of the abducens—the so-called *Syndrome Gradenigo*—has been observed as a complication of otitis media without abscess or other demonstrable cerebral lesion. I have seen several instances of this combination, in each of which the lesion was attributed to a cerebral œdema, for all of the patients showed headache, vomiting, and a low grade of choked disc. These œdemas are presumably akin to the meningitis serosa of Quincke, and in most of our cases a lumbar puncture or a subtemporal decompression has not only largely relieved the pressure symptoms, but has led to a rapid subsidence of the diplopia. The fluid in all of these cases has been sterile. It is not improbable, therefore, that disturbances of this kind may be ascribed to some swelling and œdema of the brain-stem which had brought about the same constricting effect on the part of the cerebellar arteries that we have described as existing in cases of brain tumour.

A similar explanation may account for the mysterious abducens palsies which occasionally appear a few days after the subarachnoid injection of stovaine,[1] for the profound headache which so often follows this procedure is quite possibly due to an œdema of the brain-stem. On a similar basis—namely, of transient œdemas—one is tempted to speculate on the recurrent diplopias which accompany certain ophthalmic types of recurring migraine, though it must be acknowledged that the oculomotor lesion involving either the third or sixth nerve is, like the headache, usually, though not always, confined to one side. A collection of thirty-six of these cases was made by Spicer and Ormerod [17] in 1896, and, hesitating to accept the usual functional interpretation of the symptom, they were inclined to ascribe it to some organic lesion.

[1] The subject is fully reviewed in a recent paper by Wendell Reber.

Arterial Constriction of other Cranial Nerves.

It is well known that nerves may be rendered incapable of transmitting impulses—"blocked," in other words—by relatively slight grades of constriction not necessarily sufficient to produce degenerative changes. It is well known, too, that the ocular palsies occurring in cases of brain tumour may be more or less fluctuating in their intensity—a condition of things easily accounted for on the basis of an arterial constriction which varies in degree from time to time.

A form of vascular indentation affecting the third pair has been observed in a number of the brains in our collection, many of them representing cases in which paralyses of the *Nn. oculomotorii* have proven to be "false localizing signs." It can be seen in fig. 4 that the *A. cerebelli superior* and the *A. cerebri posterior* may lie in close proximity to one another rather than at a distance, as appears in fig. 2. Inasmuch as the third nerve emerges between these two vessels it could easily become kinked or deeply indented by closely opposed arteries, particularly should any considerable change of position in the brain-stem be accompanied by such an increase in size of the subtentorial structures as would tend to put these vessels on the stretch. Though our observations on this point have been less thorough than those regarding constriction of the sixth pair, we nevertheless have encountered a number of striking illustrations. The condition is well shown in the photograph of the brain-stem of Case No. 11 (fig. 4).

In speaking of the general effects of pressure exerted on the brain and cerebral nerves by a tumour, Bruns [2] refers to the possibility that paralyses may be due, not only to direct involvement in or pressure by a growth or gumma, but also to the dislocation (Verschiebungen) of the brain itself. He quotes Wernicke, to the effect that a basal tumour on one side can dislocate the brain in such a way as to cause paralyses of the contralateral cerebral nerves, which become pressed against the sharp dural borders of their foramina of emergence, the nerves on the side of the tumour being greatly stretched at the same time. He says further:—

"Schliesslich, darauf hat Wernicke besonders hingewiesen, können durch Verschiebung der Hirnmassen Gefässe, die einen Hirnnerven umspannen, an denselben angedrückt werden und ihn schliesslich direkt durchschneiden; das ist am Olfactorius, am Tractus und Nervus opticus, am Oculomotorius und Abducens durch die entsprechenden Gefässe beobachtet—neben der Verschie-

lung der Hirnteile spielt hier allerdings wohl auch die Spannung durch den Hydrops ventriculorum eine grosse Rolle."[1]

In view of this concise statement it is possible that the association of palsies and vascular constriction is a matter of more common knowledge than I had supposed, though I have failed to find any definite note in regard to the particular form of strangulation of the abducentes which has been gone into so fully.

Instances of arterial grooving of other nerves have been given by a number of writers. Thus, Theodore Sachs [14] pointed out that a factor, at least in the optic atrophy, so often associated with a tumour of the pituitary body, may be an indentation of the optic tracts where they are crossed by the externally placed carotid or its anterior cerebral branches, his view being that the slowly-enlarging growth situated within the confined space of the chiasma pushes the nerves against the resistant vessels, which do the damage rather than the bony walls of the chamber. He pictures very clearly such a transverse grooving of the optic nerves, and has made studies of the resultant degenerations. It, however, does not appear to be a condition of such rarity as Sachs was led to believe, for the illustration accompanying his article might well serve to depict precisely the same vascular indentation observed in two of our own cases of hypophysial tumour with optic atrophy. Indeed, Sachs was not the first to make this observation, for Türck had called attention to a similar case forty years before, and Leber had pointed out that the constriction was presumably due to the fact that the tumour had put the vessels under tension, so that the intervening soft nerve-substance had suffered in consequence.

On similar grounds, Oppenheim [8] has accounted for the unusual homolateral hemiparesis and hemianalgesia observed in a case of cerebello-pontine tumour; for he found, post-mortem, that the vertebral artery on the opposite side to the lesion lay tightly stretched across the pons at the bottom of a deep groove, which suggested the likelihood of an interference with conduction from this cause rather than from pressure of the structures against the cranial base, or from stretching of the fibres. Doubtless many other similar examples of vascular constriction could be found.

[1] I have failed to trace in Wernicke's writings the original source of this statement for which Bruns gives no reference. There is an article by Otto Wernicke, of Buenos Ayres ("Ueber Lähmung der Seitwärtswender," *Arch. f. Augenh.*, 1900, xli, s. 183), describing the occurrence as rare of a double abducens palsy in a case of multiple tuberculoma seen in the clinic of Robert Wernicke. One of the lesions occupied the pons, and to this the ocular symptoms were accredited. The author acknowledges difficulty in accounting for the absence of a bilateral facial palsy, and does not believe that the usual explanation of the greater resistance of the seventh nuclei to pressure really solves the question.

Though it is desirable to lay emphasis on the cerebral nerve constrictions which may be produced by normal vessels, a word may be said in regard to the neighbourhood paralyses produced by basilar vessels tortuous and stiffened from disease. For obvious reasons these lesions have been more commonly observed than the constrictions by normal vessels, for with the former it requires no intracranial fixation of the tissues to set the lesion so that it is apparent after removal of the encephalon. Owing to the primary importance of the optic nerves and the detailed studies made by ophthalmologists in seeking for the causes of visual disturbance, we are especially familiar with the pressure effects of atheromatous vessels in the neighbourhood of the chiasma.

In his monograph on the subject, Otto [9] reported a number of instances emphasizing the mechanical effect of the dilated and stiffened vessels in pressing the nerves against some resistant structure like the optic foramen [21], and cases are recorded [1] of almost complete division of the nerve from the pressure of a sclerosed ophthalmic artery.

Similar affection of other of the cerebral nerves may readily be produced by the juxtaposition of an extensively diseased and stiffened artery. The symptoms, however, in most cases of pronounced cerebral arterio-sclerosis represent such diffuse changes on the part of the cerebrum as a whole that the possible pressure palsies of individual nerves are less clear and naturally less frequently investigated than is the case with the opticus. There are a number of brains in our series showing one or another of the cerebral nerves in dangerous proximity to atheromatous arteries, but in none of them has there been any grooving of the brain-stem by the arterio-sclerotic vessels comparable in any way to the constriction produced by normal arteries in the tumour cases. In the brains of our collection (cf. Table II.) even when the basilar, the vertebrals and their several branches, show extensive disease, the vessels, though stiffened and tortuous, usually (though not always) lie free from the pons without leaving their imprint on the nerve structures.

Thus it would seem that some enlargement of the subtentorial structures, due to a growth or to œdema which has tended to put upon the stretch the normal arteries encircling the brain-stem, is the chief element in producing the form of vascular strangulation most likely to affect the function of the sixth pair.

Conclusions.

The usual conjectures offered in explanation of the common occurrence of an internal squint in cases of brain tumour are not entirely satis-

factory, and the view is submitted that in certain cases the condition may be due to strangulation by transverse branches of the basilar artery.

This view is supported by the findings (1) that the arteries, contrary to the usual anatomical description, generally overlie the nerves; (2) that in a series of brains of tumour cases the vessels which normally encircle the brain-stem often produce a more or less deep grooving of the nervous tissues; (3) that the abducentes in many of these cases are deeply constricted: and (4) that a large percentage of the clinical histories of cases which show post-mortem a pontine grooving, with accompanying implication of the nerves, record either subjective diplopia or the actual presence of a convergent squint observed during life.

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TUMOURS OF THE THIRD VENTRICLE, WITH THE ESTABLISHMENT OF A SYMPTOM-COMPLEX.

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In a careful *résumé* of the literature the writer was struck with the lack of accurate knowledge regarding the diagnosis of tumours in the third ventricle, for, curiously enough, no attempt has been made to collect and analyse the cases bearing upon this subject. Within a few years I have seen three cases of tumour of the third ventricle, and was impressed with the possibility of making a correct diagnosis at least in some of the cases, and with this idea in view have studied all those with necropsy which could be found in the literature. Altogether I have collected twenty-seven, which, with my own, make thirty. It is, of course, probable that some have been overlooked.

These cases have been limited entirely to tumours which grew from the choroid plexus or the ependymal walls, and those which extended into the third ventricle from the surrounding structures have been excluded as well as those in which the hypophysis was involved.

Most are, clinically, of not much import, as few were well studied. In many only the post-mortem findings were given, while the cases in which both the clinical and pathological reports were complete are rare indeed.

Remarks.

The third ventricle in life contains cerebrospinal fluid and part of the choroid plexus. Most of the cerebrospinal fluid is secreted by the choroid plexus of the lateral ventricles, and the natural flow is from the lateral to the third and from the third to the fourth ventricle by means of the aqueduct of Sylvius, and then to the subarachnoid space through the foramen of Magendie. Any lesion, therefore, of the third ventricle

is liable physically to interfere with the normal flow of the fluid. Manifestly, a small tumour situated in the floor of the ventricle and not interfering either with the foramina of Monro or the aqueduct of Sylvius may not cause much obstruction, whereas a tumour so situated as to hinder either of these communications will promptly cause symptoms.

A tumour, no matter how situated, unless of small size, must cause pressure on the choroid plexus, and therefore interfere with its normal function. This in some way causes over-accumulation of fluid. According to Mott, in larger tumours it may be partially due to pressure on the veins of Galen, which interferes with the venous return from the choroid plexus and thereby causes not only accumulation of cerebrospinal fluid in the ventricles, but also serous exudation. In other words, in lesions of the third ventricle an internal hydrocephalus will necessarily be produced. This is borne out by the study of the cases in the literature, for of these, internal hydrocephalus was present in every case with the exception of three, in which it was not mentioned, but in which the observations may not have been made.

Ordinarily, in an acquired internal hydrocephalus all the ventricles are equally dilated, but it can be readily seen that in such instances as in Case 3 (Weisenburg), in which there was direct obstruction of the foramina of Monro, there may be predominant dilatation of the lateral ventricles with only little of the third.

The more direct group of symptoms which result from lesions of the third ventricle depend upon pressure on the surrounding structures. The third ventricle is surrounded on either side by the optic thalami, inferiorly by the optic chiasma and pituitary body, and superiorly by the velum interpositum which transmits the veins of Galen. Its outlets are the foramina of Monro and the aqueduct of Sylvius. When there is encroachment upon its space the increase in pressure which results is transmitted along the lines of least resistance to the other cavities through the foramina of Monro and aqueduct of Sylvius, rather than to the structures which offer solid resistance, such as the thalamus.

It is necessary, before discussing symptoms more specifically, to consider the characteristics of growths which occur in the third ventricle. As has already been mentioned, those tumours only were considered which grew either from the choroid plexus or from the ventricular walls. Their nature varied considerably, but had no direct bearing upon the symptoms, which depended entirely upon their size and situation. Manifestly a tumour of small size situated in the floor of the ventricle

Reported by	General Symptoms	Ocular Symptoms	Inco-ordination	Mental Symptoms	Other Symptoms	Post-mortem Findings
Barié. Referred to by Schuster, Paul. "Psychische Störungen bei Hirntumoren." Stuttgart, 1902, S. 168. (Unable to obtain original reference.) Woman, aged 56	Not mentioned	Not mentioned	Not mentioned	Stupidity	Paresis and anæsthesia of both extremities	Glioma of third ventricle.
Bielchowsky, Max. *Deut. Zeitschrift f. Nervenheilk.*, 1902, Bd. xxii. S. 54. Girl. aged 18	Headache, vomiting, choked disc	Paralysis of associated ocular movement and of convergence upward and downward, lateral movements being also impaired; left pupil larger than right, both pupillary reactions lost, diplopia	Cerebellar inco-ordination	Tendency to somnolence	Disturbance of hearing, slow speech, weakness in right side, increased knee-jerks	Epithelial tumour growing from the choroid plexus in the third ventricle extending into the aqueduct of Sylvius and bisecting the parts anteriorly in the cerebral peduncles compressing and pushing apart the red nuclei, compressing the left foot and the anterior and posterior corpora quadrigemina, also both pulvinars. Most of the cells of the third nuclei were destroyed. Dilatation of all the ventricles.
Birch-Hirschfeld. "Lehrbuch," 1st half, 1897, p. 230	No clinical history	.. ..	.. ..	.. .. ..	.. .. ..	Papilloma floor of third ventricle.
Bollinger, O. *Münchener med. Wochenschr.*, 1887, Bd. xxxiv, S. 789. Woman, aged 26	Recurrent headache, dizzy attacks and vomiting, choked disc	Paresis of left sixth ..	Not mentioned	Slow apathetic mentality, unconscious attacks	Not mentioned ..	Primary actino-mycosis growing from the ependymal walls of the third ventricle, compressing the optic thalamus and anteriorly the fornix and septum pallucidum. Dilatation of the ventricles.
Bristowe, J. S. *Brain*, 1883, p. 182. Boy, aged 4.	Optic neuritis, congestion of the veins in the upper eyelids	Diplopia	Not mentioned, although diagnosis was made of cerebellar tumour	Drowsiness ..	Symptoms began with tremor and loss of power in left limbs followed by similar condition in right	Tubercular mass in third ventricle involving both thalami, followed by tubercular meningitis. Small tubercular mass found on internal surface of left occipital lobe. Dilatation of lateral ventricles.
Calonzi, A. *Il Policlinico*, 1908, vol. xv, p. 130. Boy, aged 12	Headache, nausea, vomiting, vertigo, choked disc	Paresis of associated upward ocular movement and of both external recti	Cerebellar gait	Not mentioned ..	Reflexes normal	Cystic tumour growing from the choroid plexus, extending to the pituitary body and into the aqueduct of Sylvius. Dilatation of lateral ventricles.
Cornil, V., and Ran.	No clinical	.. ..	.. ..	.. ..	.. ..	Papilloma growing from the

aged 12	Vertigo, choked disc	external recti				The aqueduct of Sylvius. Dilatation of lateral ventricles.
Cornil, V., and Ranvier, L. "Manual of Pathological Histology," 1880, Phila., Trans., vol. i, p. 378.	No clinical history appended	.. .. ..	.. ..	.. ..	.. ..	Papilloma growing from the ependyma of the third ventricle, projecting into the lateral ventricles through the foramen of Monro.
Falkson, R. *Virchow's Archiv.*, 1879, Bd. lxxv, S. 550. Boy, aged 16	Failing vision, no choked disc	Diplopia	Not mentioned	Stupidity, loss of memory	Not mentioned ..	Chondro-cysto-sarcoma growing from ependymal walls.
Finkelnburg, R. *Deut. Zeitschrift f. Nervenheilk.*, 1902. Bd. xxi, S. 460	Headache, choked disc, nausea, vomiting, vertigo. Disappearance for six months with recurrence	Right pupil larger than left with prompt reactions, paresis of external muscles of right third with the exception of levator and of right fourth and both sixth. These symptoms almost disappeared for six months with sudden recurrence of irregular pupils and impaired reactions, bilateral ptosis, weakness of all the muscles of both thirds	Cerebellar gait	Dull, apathetic, tendency to somnolence	Weakness of left lower seventh and twelfth and of left limbs with increased reflexes, this almost disappearing for six months and then reappearing	Cystic glioma of the third ventricle affecting right thalamus, tumour extending into the aqueduct of Sylvius and compressing the anterior corpora quadrigemina and posterior part of the peduncle and pons. Dilatation of lateral ventricles. No microscopic examination of brain.
Dutkin, Esther. *Freiburg i/B.*, 1905, I. D. (Unable to obtain original.) Boy, aged 16	Periodic headache and vomiting, later disturbance of vision and optic atrophy	Not mentioned	Not mentioned	Normal	Reflexes increased	Cholesteatoma in third ventricle. Internal hydrocephalus.
Hennenberg, R. *Charité Annalen*, 1902, Bd. xxvii, S. 516. Case 2. Man, aged 50	Headache, margins of discs blurred	Left pupil larger than right, lost reactions	Normal ..	Dementia ..	Tongue deviated to the left, reflexes normal	Papilloma growing from the choroid plexus of the third ventricle, extending into the aqueduct of Sylvius which was much dilated. The tumour grew in the midline and separated the red nuclei, compressing them as well as the anterior corpora quadrigemina. In the left third nuclei was a fresh hæmorrhage and the ganglion cells were diseased. The lateral ventricles were only slightly dilated.

Reported by	General Symptoms	Ocular Symptoms	Incoordination	Mental Symptoms	Other Symptoms	Post-mortem Findings
Kauffman, O. J. *Birmingham Medical Review*, 1905, p. 231. Man, aged 50	Headache and dizziness	Blindness from birth ..	Not mentioned	Not mentioned ..	Not mentioned ..	Myxosarcoma of third ventricle.
Meyer, E. *Archiv. f. Psychiatrie u. Nervenkrankheiten*, 1899, Bd. xxxii, S. 320. Man, aged 58	Headache, vertigo, vomiting, later optic neuritis	Reaction of left pupil diminished	Cerebellar gait. Tendency to fall backward and to the right	Loss of memory and orientation of time and place, false reminiscences and fabrications, resembling Korsakoff's psychosis	Limbs weak, left more than right. Knee-jerks increased	Sarcomatous tumour of the third ventricle projecting into the lateral through the foramen of Monro, with metastasis into the floor of the fourth ventricle, the aqueduct being normal. All the ventricles were dilated. Hypophysis normal. No microscopic examination of brain.
Mott, F. W., and Barrett, J. O. W. *Archives of Neurology*, 1909, vol. i, p. 417. Case 1. Man, aged 34	Headache, nausea, vomiting, dizziness, choked disc	Periodic attacks of double vision, ptosis, stupor lasting about a week, followed after some years by sluggish pupils and left ptosis	Cerebellar gait more to the left	Periodic attacks of drowsiness and stupor, tendency to somnolence, later impairment of memory, tremors in face and tongue, leading to diagnosis of paretic dementia	Weakness especially in left limbs with increased reflexes	Colloid cyst the size of a Barcelona nut situated above the optic tract, its centre corresponding to the termination of the infundibulum, attached to the choroid plexus from which it grew. Dilatation of lateral and third ventricles. Hypophysis not examined.
Case 2. Man, aged 39	Was insufficiently studied	Pupils dilated, reactions sluggish	Unsteady gait	Regarded as an epileptic and general paralytic	Epileptic attacks, increased reflexes	Mycotic infection setting up new growth in body of third ventricle by irritation. Dilatation of all ventricles.
Case 3. Man, aged 28	Insufficiently studied, supposed to have had headache, nausea and vomiting	No note made	Not mentioned	Diagnosed as mania with suicidal tendencies. Was very drowsy	.. .. ..	Epidermoid cyst with numerous small cysts in third ventricle. Hypophysis not examined. Dilatation of all ventricles.
Nothnagel, H. *Wiener med. Presse*, 1889, Bd. xxx, S. 89. Boy, aged 15	Headache, nausea, vomiting, vertigo	Protrusion of both eyeballs, paralysis of associated ocular movement upward and to the left, paresis of third and sixth nerves, pupils large	Cerebellar inco-ordination	Not mentioned ..	Not mentioned ..	Epithelial tumour growing from the choroid plexus of the third ventricle into the aqueduct of Sylvius and compressing the anterior corpora quadrigemina.

		and to the left, paresis of third and sixth nerves, pupils large					rigemina.
Russell. *Med. Times and Gaz.*, 1873, vol. i. p. 522. Boy, aged 15	Not mentioned	Not mentioned	Not mentioned	Not mentioned ..	Fits and tremors	Sarcoma base of third ventricle attached to the optic commissure. Dilatation of lateral ventricle.	
Sawyer. Quoted by E. Meyer, *Archiv. f. Psychiatrie u. Nervenheilkunde*, 1889, Bd xxxii. Man, aged 29	Headache, vomiting	Not mentioned	Ataxic gait ..	Memory poor, speech like general paresis	.. .. ..	Echinococcus cyst in floor of third ventricle with hydrocephalus.	
Saxer, F. *Zeigler's Beiträge*, 1902, Bd. xxxii, S. 333. Man, aged 46	History is very poor	.. ..	.. ..	.. .. ..	.. .. ..	Epithelial tumour growing from the epithelial lining of the infundibulum, filling up the whole ventricle. Hypophysis normal. Internal hydrocephalus.	
Selke, Walter. "Inaug. Diss.," Königsburg, 1891. Woman, aged 42	Headache, nausea, vomiting, vertigo, loss of vision, choked disc	Pupils wide and reactionless, attacks of unconsciousness in which there was deviation of the eyes to the right	Uncertain gait	Attacks of unconsciousness which lasted at times eight days	Twitching in left limbs followed by left hemiplegia	Papilloma growing from the choroid plexus causing pressure on the optic chiasm. Dilatation of lateral ventricle. No microscopic examination of brain.	
Scholz, Fritz. *Virchow's Archiv.*, 1906, Bd. clxxxiv, S. 255. Man, aged 30	Headache, vomiting, choked disc, blindness	Pupils equal, normal reactions	Uncertain gait	Somnolence ..	Power in left limbs diminished with diminished reflexes	Cholesteatoma of epidermoid origin, compressing surrounding structures and extending into the lateral ventricles. Dilatation of lateral ventricles. No microscopic examination.	
Tannenheim, E. *Wiener klin. Wochenschr.*, 1897, S. 494. Man, aged 40	Headache, vomiting, choked disc	Not mentioned	Not mentioned	Stuporous condition	Increased reflexes	Dermoid cyst in third ventricle. Dilatation of lateral ventricles.	
Wallman, Heinrich. *Virchow's Archiv.*, 1858, Bd. xiv, S. 385. Man, aged 45	Not mentioned	"Eyes weak"	Uncertain gait	Memory poor ..	Weakness in feet	Colloid cyst the size of a walnut in the third ventricle attached to posterior commissure. Also small lipoma of right lateral ventricle. Internal hydrocephalus.	
Williamson, R. T. *Medical Chronicle*, 1905, p. 76. Boy, aged 12	Marked symptoms of intracranial pressure, especially headache and choked disc	Paralysis of right external and internal rectus, movement of right eyeball upwards and downwards impaired, movements of left eye impaired to the right	Tendency to fall to the right	Drowsy and comatose	Left sensory fifth involved, knee-jerks absent	Round-cell sarcoma growing from the choroid plexus of the third ventricle the size of a small hen's egg, with dilatation of the lateral ventricles. No microscopic examination of brain.	

Reported by	General Symptoms	Ocular Symptoms	Inco-ordination	Mental Symptoms	Other Symptoms	Post-mortem Findings
Wilson, S. A. K. *Brain*, 1906, vol. xxix, p. 527. Case 2. Man, aged 42	Headache, vomiting, dizziness, choked disc	Left pupil larger than right, reactions slow, slight double ptosis, divergent strabismus, periodic ectopia pupillæ, later upward movements defective, on convergence only the right eye moved in, conjugate deviation to the left limited, with increased ptosis. Slight proptosis	Not mentioned	Dreamy condition	Occasional attacks of unconsciousness during which towards the last the limbs were flaccid	Colloid tumour in third ventricle attached to the ependymal wall, flattening the pons and dilating the aqueduct of Sylvius. No microscopic examination.
Woodbury, F. *Amer. Journ. Med. Sci.*, July-Oct. 1878, p. 128. Man, aged 45	Headache, vertigo, negative eye grounds	Divergent squint and diplopia	Cerebellar gait, backward staggering	Mental lethargy	Weakness in limbs, tinnitus	Gliosarcoma in third ventricle extending into and blocking up the aqueduct of Sylvius. Dilatation of lateral and fourth ventricle. Small area of softening superior surface of cerebellum.
Weisenburg, T. H. Case 1. Girl, aged 17	Headache, morning nausea and vomiting, dizziness, marked choked disc, failing vision	Weakness of both external recti, paresis of associated ocular movement and of convergence upwards and to a less extent to the left, pupils dilated and reactions slow. Slight proptosis	Cerebellar gait, incoordination of limbs, deviation of head to left	Memory impaired, somewhat stupid	General weakness of limbs, loss of all reflexes with retention of the plantar, weakness in lower part of left face	Large vascular glioma growing from the ependymal walls extending into the lateral ventricle and aqueduct of Sylvius, compressing the surrounding structures. Continuous with optic thalamus. Growth bisected almost completely the cerebral peduncle anterior to the aqueduct, destroying almost wholly the third nuclei, and compressing the red nuclei, and destroying the commissural fibres. Dilatation of lateral ventricles.

Case 2.	Girl, aged 20	Failing vision, headache, nausea, vomiting, dizziness, choked disc	Great protrusion on both eyeballs with downward deviation, ptosis of both upper lids with dilatation of veins, paralysis of associated ocular movement and of convergence upward, less to the right and left and only a little downwards. Pupils large and reactionless	Early cerebellar gait. Questionable ataxia of limbs	Stupid apathetic condition	Because of involvement of the thalamus had central pains and vasomotor and trophic phenomena, involuntary yelling and deviation of the body to the opposite side. Besides, had weakness in right motor fifth, lower seventh and tongue, due to involvement of knee of internal capsule. Loss of reflexes with the exception of the plantar	Glioma growing from the ependymal walls of the left lateral ventricle, extending into the third through the foramen of Monro and occupying the anterior portion of the third ventricle. The tumour extended into the left thalamus, destroying most of the anterior nuclei. Dilatation of lateral ventricles.
Case 3.	Man, aged 27	Headache, nausea, vomiting, dizziness, choked disc	Loss of vision was first noticed when head was inclined to one side. When body was held rigid with head tilted back vision was best. When head was tilted forward vision would gradually fade and would return on resuming an erect posture. Failing vision was accompanied by marked headache and dizziness	None ..	None	None	Colloid cyst the size of a small marble attached to the choroid plexus in the upper anterior portion of the third ventricle posterior to the middle commissure, so situated that it could close the foramen of Monro. Marked dilatation of lateral ventricles with little of third.

Dr. Arthur Hall, of London, reported two cases of tumour of the third ventricle before the Pathological Society, but I have been unable to obtain a report of them.

would cause little pressure upon the surrounding structures, whereas a similar growth near the Sylvian aqueduct or the foramina of Monro might cause in the former the symptoms of pressure upon the oculomotor nucleus and superior cerebellar peduncle, and in the latter, besides dilatation of the ventricles, there may be no other symptoms, with the exception that if the tumour is small and movable, such as Case 3 (Weisenburg), there would be a closure or opening of the communications between the lateral and third ventricles by alterations in the position of the head; but this, of course, is exceptional.

The larger growths may be limited to the ventricle, or may grow by extension into the surrounding cavities. The tendency is nearly always to grow from the third to the fourth ventricle, through the aqueduct of Sylvius, in the direction of the flow of the fluid, rather than into the lateral ventricle. Of the collected cases, three only extended into the lateral ventricles. Here the tumour did not actually enter the ventricles, but only projected through the foramina, owing principally to its situation in the anterior portion of the cavity. But in seven cases there was extension into the aqueduct of Sylvius. It is interesting to observe that in three of these (Weisenburg, Case 1, Bielchowsky, and Henneberg) the tumour grew ventrally from the aqueduct into the cerebral peduncle, almost bisecting it destroying the oculomotor nuclei, compressing and separating the red nuclei; while in three other cases (Calonzi, Nothnagel, and Woodbury) the posterior part of the cerebral peduncle and pons was much compressed, with no forward extension. In Finkelnburg's case the growth affected the posterior part of the peduncle and pons, especially the right, and compressed the anterior corpora quadrigemina. In this connexion Meyer's case is interesting because there was metastasis to the fourth ventricle, the aqueduct escaping. In another case reported by Finkelnburg (Case 8) a sarcoma grew in the third ventricle, and extended into the lateral ventricles and aqueduct of Sylvius as well as the surrounding structures. The hypophysis was affected.

It can be seen, then, that, so far as the symptomatology is concerned, it is necessary to make three distinct classifications—(1) those cases in which a tumour of moderate size is situated in the floor of the third ventricle, and in which there is no extension into the foramina of Monro or aqueduct of Sylvius; (2) small tumours so situated as to obstruct the foramina of Monro, and whose position can be changed by deviation of the head; and, lastly, (3) tumours, whether large or small, which either extend into the aqueduct of Sylvius, affecting the surrounding struc-

tures by direct extension or pressure, or those in which the posterior portions of the cerebral peduncle and pons are compressed either by direct pressure or dilatation of the aqueduct of Sylvius. These will be discussed in order.

First Class.

Those tumours which enter into this classification do not offer specific symptoms, but depend upon those of pressure on the surrounding structures. As has already been mentioned, all growths of the third ventricle cause internal hydrocephalus, and therefore its symptoms of this condition will be discussed first, as they apply to all the classifications.

In the collected cases they consisted principally of headache and choked disc, and to a less degree of nausea and vomiting and of dizziness. In nearly all there was also cerebellar inco-ordination. As can be seen, the general symptoms of increased intracranial pressure were present which are produced either by tumours of the posterior cranial fossa or by internal hydrocephalus, the differential diagnosis between them, as is well known, being sometimes impossible.

It might be expected that large tumours in this area would cause thalamic symptoms, but, as has already been mentioned, this does not often occur, and even if there is general pressure on the thalamus no specific symptoms would be manifested excepting in those cases in which there is direct destruction, as in Case 2 (Weisenburg), in which there were central pains, vasomotor and trophic disturbances, principally on the side opposite the lesion, involuntary howling and deviation of the body to the opposite side, this being the beginning of a progression movement. In only three other cases of the thirty was the thalamus affected, but there were no symptoms mentioned referable to it.

In tumours of large size some indirect pressure is nearly always exerted on the internal capsule of one or both sides, and there should be present corresponding paresis of the limbs; but, as is well known, this sometimes also occurs in internal hydrocephalus, and yet it should be more common in large tumours of the third ventricle, because there is more likely to be also pressure on the internal capsule. It should be remembered that in internal hydrocephalus the paresis may result from cortical pressure. It can be seen, then, that the combination of these conditions should more readily produce paresis of the limbs. Of the collected cases, in seven there was paresis of one side and in five of both, and, while this occurred in twelve only of the thirty cases, in those

in which it was absent the tumours were so small that no pressure was exerted upon the surrounding structures.

The character of the paresis is that seen in internal hydrocephalus—that is, a paresis without a well-defined paralysis, there being nearly always spasticity of the limbs, although they may be flaccid. The reflexes nearly always are increased, but sometimes may be normal or diminished.

Somehow the impression has gained ground that lesions of the third ventricle may cause mental symptoms, such as a drowsy, apathetic dull mentality, and often greater impairment. In a number of the collected cases the mental condition was that of general paresis (Henneberg and Mott and Barratt, Cases 1 and 2), while Meyer's case presented the symptoms of Korsakoff's psychosis. In sixteen there was present either a dull, stupid mentality or a decided tendency to somnolence or drowsiness; while in Case 1, Mott and Barratt, there were "recurrent attacks of drowsy stupor, dementia and slowness of thought, speech and movement, and tremor of the tongue and face, so that at one time the patient simulated an epileptic, at another a general paralytic, and then again became almost sane." Mott thought that the mental symptoms were the result of compression of the cortex by the internal hydrocephalus, and explained the recurrences by the escape of fluid from the lateral ventricles, thereby relieving the cerebral compression and removing the cortical anæmia.

Purves Stewart [3], in discussing the causes of drowsiness, believes that it might result from "upward pressure on the floor of the third ventricle whether this be by direct pressure on the ventricle and Sylvian aqueduct, or whether, as Franceschi suggests, it be the result of cerebral anæmia from compression of the arteries of the circle of Willis between the upper surface of the growth and the under surface of the brain. The curious intermissions in the drowsiness might be explained on either hypothesis, either by variations in the tension of a cystic tumour or by alterations in the direction of the growth with relation to the superjacent vessels."

John Turner [4] disagrees with Stewart's conclusions, and believes that the drowsiness is due to a cerebral œdema. It has been claimed by some that coma may be produced by a deprivation of the secretion of the hypophysis. This does not enter into the discussion of tumours within the third ventricle, because all those in which the hypophysis was involved were rejected. Again, in one of Stewart's cases in which the hypophysis was entirely destroyed there was no stupor.

It is possible that the drowsy, somnolent condition which is so frequent an accompaniment of tumours in the third ventricle is brought about by impairment of the normal function of the cortex, the result of pressure against the skull through dilatation of the lateral ventricles, and is not a specific symptom resulting from lesions of the third ventricle.

Second Class.

It would seem hardly necessary to make this classification, inasmuch as there is only one tumour (Case 3, Weisenburg) which enters into it. The patient, a man, first noticed failure of vision on bending his head to one side, and, with the accompanying symptoms of intracranial pressure, had most vision and less general discomfort when his body was erect, with the head tilted backward, while any deviation from this posture caused increase in his headache and nausea, with total loss of vision, which would return on resuming the primary position. At necropsy a tumour was found attached to the choroid plexus, the size of a small marble, situated in the upper anterior portion of the third ventricle, in front of the middle commissure closing the foramina of Monro. It was so loosely attached that undoubtedly deviation of the head might have caused the growth to be moved from its position.

It is rather difficult to explain what occurred in this case. It is apparent from its situation that there was a gradual closure of the foramina of Monro, which produced a dilatation of the lateral ventricles, the third being only slightly dilated. The patient felt most comfortable and had most sight with his head tilted backward, probably because in this position there was established free communication between the lateral and third ventricles. While it is explicable that the headache and nausea were increased by the closure of the foramina of Monro, which occurred when the head was tilted forward, inasmuch as in this position the tumour effectually closed up the foramina of Monro and increased the pressure in the lateral ventricles, it is rather difficult to understand how this would cause total loss of vision, and yet this is the only explanation possible, inasmuch as tilting the head backward caused return of sight.

If, then, an increase in the intracranial pressure in the lateral ventricles can cause headache and nausea evidently through pressure on the cortex, just so there might have been produced by pressure on the occipital cortex failure of vision. It must be remembered that the patient had choked disc and only one eye, the other having been removed because of an injury, and that sight at best was impaired.

Alterations in the general symptoms of tumour, particularly vomiting and dizziness, have been observed, especially in cerebellar tumours, and less so in tumours of other areas. The usual explanation given is that in certain positions of the head there is interference or closure of the communication between the ventricles, especially in the aqueduct, this causing increase in intracranial pressure; but I have been unable to find an instance in which vision was similarly affected as in the case above mentioned.

Third Class.

It is upon the cases which come into this classification that a definite diagnosis can be made, and in which it is possible to establish a fairly well recognizable symptom-complex. The diagnosis is dependent entirely upon the fact that there is involvement either directly or by pressure of the oculomotor nuclei, the red nuclei or the superior cerebellar peduncles, and pressure or destruction of the posterior longitudinal bundle, or the intercommunicating fibres between the third nuclei. In some of the cases which come under this classification the anterior corpora quadrigemina were also pressed upon.

In the *résumé* of the reported cases it will be seen that most of the observations were very poor, and that in many of the cases in which the oculomotor nuclei were directly affected no mention whatever is made of ocular symptoms. So far as paralysis of associated ocular movement is concerned, there are very few observations indeed, especially in the older reported cases when the causes of "Blicklähmung" were not well recognized.

Within a few years it has been definitely established that lesions near the aqueduct of Sylvius cause paralysis of associated ocular movement upward and more rarely downward, and that impairment of lateral deviation is caused by affection of the posterior longitudinal bundles.

Of the instances of third ventricle tumours in which there occurred "Blicklähmung" four types of lesions can be differentiated:—

(1) Those in which the lesion was above the aqueduct of Sylvius, such as Case 2 (Weisenburg), in which the tumour was in the upper anterior portion of the third ventricle, and in which there was no direct involvement of the oculomotor nuclei. Microscopically, however, the cells were distinctly diseased, and there was besides a definite internal hydrocephalus with dilatation of the aqueduct. It is probable that the paralyses of associated ocular movement in this case were the result of disease of the third nuclei from pressure. In this case, besides para-

lysis of upward movement, there was impairment to the right and left and slightly downwards.

(2) In the second type the pressure is more direct, such as in Wilson's case, in which the tumour did not extend into the aqueduct, but compressed directly the upper part of the peduncle and pons. In this case there was weakness of upward associated ocular movement and of conjugate deviation to the left.

(3) In the third type are those cases in which, while the tumour extended into the aqueduct, the surrounding structures were involved by pressure, there being no extension. Such were the cases of Nothnagel and Calonzi. In the former there was associated weakness upward and to the left, and in the latter weakness only in an upward direction. In Woodbury's case the tumour extended into the aqueduct, but the only note made is that there was divergent squint and diplopia. Considering, however, the early report of the case (1878) "Blicklähmung" might have been overlooked.

(4) In the fourth type are those cases in which the growth extended into the aqueduct of Sylvius and from it ventrally, bisecting the peduncle and causing almost complete destruction of the oculomotor nuclei. This was true in the cases of Bielchowsky, Weisenburg (Case 1), Henneberg, and Finkelnburg. In the first there was paralysis of associated ocular movement upwards and downwards, lateral movements being also impaired. In my case there was paralysis of associated ocular movement and of convergence upwards and less to the left, while in Henneberg's case there is no mention of this symptom, although the nuclei were distinctly diseased, as were also the red nuclei, no mention being made also of inco-ordination. In Finkelnburg's case the statement is made that all the muscles of both oculomotor nuclei were affected, but that, on account of the mental condition, closer observation was impossible.

It is evident, then, that in lesions of the third ventricle, whether because of direct or indirect pressure from above or by direct affection of the oculomotor area, there occurs paralysis of associated ocular movement, predominantly in an upward direction and more rarely downward and to either side. With the exception of Bielchowsky's case and Weisenburg's Case 2 there was no mention of paresis of downward associated ocular movement. In all the other cases, in association with paralysis of upward movement, there was impairment of associated ocular movement either to one side or both sides.

With "Blicklähmung" there should be paralysis of convergence in

the same directions. With the exception of my own cases, however, this was mentioned only by Bielchowsky. The question of convergence, while apparently simple, has by no means been threshed out. By most text-books and writers on this subject one is left under the impression that convergence is possible only in a median direction, and the statement is constantly made that there is paralysis of associated ocular movement, but that convergence in the same direction is possible, which is manifestly contradictory, for converging movements are in their very essence associated. The truth of the matter is that convergence is normally possible in all directions, and the writer is of the opinion that all ocular movements are in convergence.

Of the other ocular symptoms which occur in such lesions the condition of the sight and pupils can be dismissed, for the former is nearly always impaired because of the choked disc, the condition of the pupils being dependent upon the same condition as affection of the third nerve nuclei.

In those cases in which there is direct affection of the oculomotor nucleus there are, of course, oculomotor palsies. This was true in the case of Nothnagel, Finkelnburg, Wilson, Bielchowsky, and Weisenburg (Case 2), in which were present ptosis and pupillary irregularities only.

Protrusion of the eyeballs was present in four cases (Nothnagel, Wilson, and Weisenburg, Cases 1 and 2). In another communication I have discussed the occurrence of exophthalmos in brain tumour, and have come to the conclusion that it occurs only in those cases in which there is either direct pressure on the cavernous sinus or the veins of Galen, this leading either to stasis of the vessels of the orbits or thrombosis of the cavernous sinus. It should be expected then that large tumours of the third ventricle should occasionally cause protrusion of the eyeballs because pressure may be exerted upon both structures.

In a number of cases there was present paralysis of the sixth nerve either on one or both sides, this being presumably due to pressure.

A most constant symptom, and one which was present in fifteen of the cases, was ataxia, which was always of the cerebellar type, the description of the gait being drunken or ataxic, there being staggering to either side and sometimes forward and backward. Besides there was mentioned in the majority of cases ataxia of the limbs. These can be undoubtedly accounted for either by a direct involvement of the red nuclei or the superior cerebellar peduncles, or by pressure upon the same structures due to dilatation of the third ventricle, or of the aqueduct of Sylvius.

The occurrence of cerebellar ataxia in internal hydrocephalus is well known, and the literature is replete with cases in which clinically the diagnosis was made of cerebellar tumour, but which pathologically demonstrated dilatation of the ventricles. The ataxia in these cases may be due to pressure on the cerebellum brought about by direct dilatation of the fourth ventricle or of the posterior horns of the lateral ventricles, but most commonly is caused by pressure upon the red nuclei or superior cerebellar peduncles. This no doubt accounts for the frequent presence of this symptom, for internal hydrocephalus was present in all but three cases in which accurate observations were not made. The conclusion, then, can be drawn that ataxia of the cerebellar type should be present in tumours of the third ventricle.

Besides the more specific localizing symptoms of "Blicklähmung" and ataxia, there were present in the cases which enter into this classification the general symptoms which have already been enumerated under the first class—that is, of increased intracranial pressure and of pressure upon the surrounding structures, for most of these tumours were of considerable size.

The symptom-complex which has been enumerated as resulting from lesions of the third ventricle which encroach upon the structures surrounding the aqueduct of Sylvius may sometimes result from lesions of this part or of the pineal gland, and yet it is possible to make a differential diagnosis.

In lesions of the pineal gland, as mentioned by Frankl-Hochwart [1] and Howell [2], there may be besides the general symptoms of tumour, "Blicklähmung," pupillary disturbances and ataxia, abnormal growth in length, unusual increase of hair, adiposity, premature sexual development, and mental precosity, none of which occur in lesions of the third ventricle. Again, as emphasized by Howell, paresis of the limbs is uncommon in tumours of the pineal gland, although internal hydrocephalus is not infrequent.

The differentiation from tumours of the anterior corpora quadrigemina is more difficult, because its symptoms are dependent upon pressure on the oculomotor nuclei, and less so on the red nuclei or superior cerebellar peduncles. The diagnosis would largely depend upon the fact that third ventricular tumours cause more symptoms because of their tendency to affect a larger number of structures; there is nearly always paresis of the limbs on one or both sides, and ataxia, the former being uncommon in tumours of the anterior corpora quadrigemina. Again, in disease of the anterior corpora quadrigemina there

would be a greater tendency to disturbance in hearing, because of affection of the posterior corpora quadrigemina.

The conclusions drawn from the study of the cases of third ventricular tumours are:—

(1) Tumours within the third ventricle growing either from the ependymal walls or the choroid plexus are uncommon; thirty cases with necropsy have been reported altogether.

(2) Their nature varies, and has no direct bearing on the symptoms.

(3) Tumours within the third ventricle have a tendency to grow in the direction of the flow of the cerebrospinal fluid—that is, towards the fourth ventricle through the aqueduct of Sylvius.

(4) Internal hydrocephalus nearly always occurs, either because of interference with the flow of the fluid or the function of the choroid plexus.

(5) Growths within the third ventricle do not cause specific symptoms, and their recognition depends entirely upon the symptoms of pressure on the surrounding structures. Three divisions can be made:—

(*a*) Tumours of moderate size limited to the third ventricle, presenting the symptoms of internal hydrocephalus, with the addition that there is nearly always paresis of the limbs of one or both sides, and possibly affection of the thalamus.

(*b*) Tumours so situated as to obstruct the foramina of Monro, cause dilatation of the lateral ventricles, and their position can be changed by deviation of the head, producing variation in the symptoms.

(*c*) Tumours which attack the parts surrounding the aqueduct of Sylvius either by direct pressure or after extension into the aqueduct by growth of the tumour ventrally, cause the following symptoms: Paralysis of associated ocular movement and of convergence upwards and, less commonly, to either side and downwards, ataxia of the cerebellar type, as shown in the gait, station or voluntary movements of the limbs, at times ptosis of the upper lids and protrusion of either one or both eyeballs, generally large pupils with impaired reactions, paresis of the limbs of one or both sides, either with increased, normal, or diminished reflexes, and the general symptoms of tumour, as headache, choked disc, nausea, vomiting, and vertigo.

(6) Lesions of the third ventricle do not cause specific mental symptoms; the occurrence of mental symptoms is dependent upon the compression of the cortex against the skull, resulting from internal hydrocephalus.

The histories of the cases are as follows:

CASE 1.—Mary C., 17 years old. Admitted to the Medico-Chirurgical Hospital, June 29, 1908. Her family and previous history were unimportant. Six months before she came under observation her menses ceased, but returned again about three months afterwards, when she first began to complain of weekly headaches, frontal in character. Six weeks later, or about two months before she was examined, the headache became constant, appearing in the morning and lasting all day. During her whole history this pain was of the same intensity, the patient describing it as a sharp and then again as a dull pain over the right side of the head and neck, right eye, and sometimes over the left. Coincident with the headache she felt sick, this appearing early in the morning and lasting until about noon. There was at the same time a tendency to vomit, but with two exceptions she never vomited.

Dizziness did not become apparent until three weeks before she came under observation, or about two months after the onset of the headache and nausea. She could not describe it any more than when she got up in the morning her head felt light and dizzy, but there was no distinct history of swaying, either of self or of objects. She was able to walk up to this time, and then noticed on arising that she would stagger as if drunk. She thought that at the same time her limbs became weak, but she meant by this that she would stagger and fall because of the ataxia rather than the result of any weakness in her limbs.

Her sight became poor, but the time of its onset could not be recalled. She thought it might have been six weeks before she came under observation. Coincidentally with the staggering she began to see double, objects appearing side by side, but not persistently. She never fainted or suffered from roaring in her ears, and hearing was good. There had never been disturbance of bladder and rectal functions. Her mentality was impaired, for it was only after repeated questioning that the history was obtained. From the very onset of the headache the patient inclined her head to the left, sometimes the ear almost approximating the left shoulder. This she did because, in this position, the intensity of the pain was diminished.

When admitted to the hospital, examination demonstrated a girl who, when lying in bed, had a tendency to incline her head to the left, this becoming much more apparent when she got up and attempted to walk. Her gait was distinctly cerebellar, walking with her feet wide apart with a tendency to stagger, especially to the left. Closing the eyes had no influence upon the ataxia. There was no tenderness to pressure anywhere over the head, but she seemed to wince a little when pressure was applied over the right cerebellar area.

Ophthalmoscopic examination by Dr. L. Webster Fox demonstrated a choked disc on both sides of about six dioptres. The pupils were dilated, and their reactions both to light and movement impaired. Vision was greatly diminished. There was a little bulging of the eyeballs. Ocular movements showed a weakness of both external recti, especially to the left. When asked to look upward there was a decided hesitancy in the beginning of the movement and the range was impaired, especially of the left eye, in which there

was present an irregular rotatory jerking. The paresis of associated ocular movement upward, however, was not complete. There was no hesitancy or jerking of the eyeballs when looking to the right, but when attempting to look to the left a rotatory movement of the left eye occurred and there seemed to be lagging of the left eyeball. Downward movement was well performed. When following a finger the same hesitancy and paresis in upward associated movement was noticed, as well as to the left. She could converge well downwards and in the median line, but when following a finger in the median line in an upward direction there was present a corkscrew-like movement of the eyeballs, the range becoming wider as the upper plane was reached, the left eye taking greater part in the movement. The same corkscrew-like movement was present when convergence was attempted from above downward, the range of the movement becoming less. Upward convergence could not be accomplished. With one eye closed both the voluntary and involuntary movements of the other eyeball seemed similar to when both eyes were exposed.

The brow could be wrinkled normally on both sides, but when the mouth was opened there was a distinct drooping on the left side. The patient could, however, wink the eye and show her teeth almost equally well on both sides. The masseters contracted well on either side, and the jaw opened in the median line. The tongue was protruded straight, there being no tremor or atrophy.

The upper limbs were well formed, there being no atrophy anywhere. Their tonicity was about normal. The grip in both hands was good, it being perhaps better on the right, the patient being right-handed. The resistance to passive movement of the upper limbs was good and about equal on both sides. The biceps and triceps reflexes were absent. Finger-to-nose test demonstrated ataxia, a little more on the left. Supination and pronation of the forearms soon showed a lagging of movement, the left arm becoming inco-ordinate first.

The lower limbs were well formed, there being no atrophy anywhere. Their tonicity was normal, and power was not at all diminished. The patellar and Achillis jerks on both sides were absent, even on reinforcement. Plantar irritation caused flexion of the toes on both sides. There was no disturbance of the sense of position or of movement, but the heel-to-knee test demonstrated ataxia of both lower limbs, distinctly more so of the left. Closure of the eyes did not increase the ataxia. Sensation for touch and pain was everywhere normal.

An exploratory operation was performed by Dr. Stillwell Burns several days after she came under observation. The left cerebellar area was exposed, but nothing was found. The patient did not do well under anæsthesia, and never regained consciousness, dying several hours after removal from the operating table. The autopsy was performed the following day, and the brain only removed.

Macroscopic Examination.

The tumour is entirely in the third ventricle and is in the shape of a lobulated egg, its anterior part projecting into the lateral ventricles through

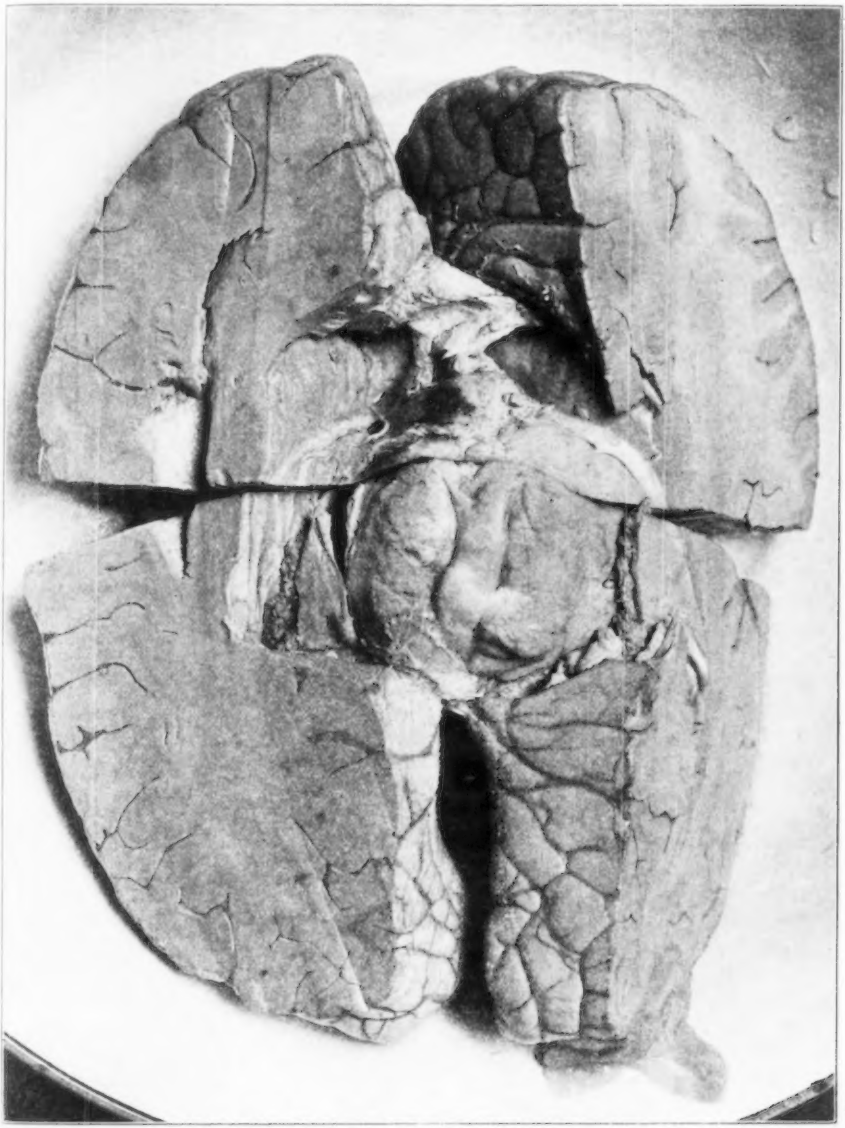

FIG. 1.

Case 1.—Tumour filling up the entire third ventricle and projecting into the lateral through the foramina of Monro.

the interventricular foramina, pushing the fornix and the lamina lucidum forward and more to the right, separating widely the caudate nuclei.

Posteriorly the tumour is round and extends by means of the aqueduct of Sylvius into the anterior part of the cerebral peduncle and the uppermost portion of the pons. The lateral ventricles are much dilated.

Inferiorly it extends to the base, dilating the infundibulum. The optic chiasma is considerably compressed. The mass of the growth leans more over to the right, compressing on either side the optic thalamus, and is continuous in part with the right. In the upper level the tumour is attached to the ventricular walls on either side, but is free below. Laterally the growth compresses, besides the thalami, the lenticular nuclei more on the right, and to some extent also the fibres of the right internal capsule. It measures $2\frac{1}{4}$ in. in length, $1\frac{1}{4}$ in. in depth, and $1\frac{1}{2}$ in. from side to side. The hypophysis is normal.

Microscopic Examination.

The *tumour* is uniform in its growth and is rich in cells which are tightly packed together, especially in its border. They are round, oval, or spindle shaped, and by the hematoxylin-eosin stain are granular in appearance. The nuclei are large, and in relation to the cell are surrounded by little protoplasm. There is very little intracellular substance, which by the neuroglia stain shows no fibres. The growth is very vascular, especially in its centre, and numerous widely dilated capillaries full of red cells and pigment are present. Occasionally the walls of these capillaries are ruptured, causing an exudation of blood-cells into the surrounding tumour tissue. In the inferior portion the outline of the growth is definite and the ependymal cells of the walls of the third ventricle can be traced both inferiorly and laterally. In its upper level the growth is continuous on each side with the ependymal walls and on the right with the thalamus. The nerve cells in both thalami are slightly degenerated and the internal capsule on the right side is slightly displaced, although no degeneration is found.

Cerebral peduncle and pons.—Cross-sections show that the tumour has extended into and obliterated the aqueduct of Sylvius, completely filling it up, extending in a wedge-shaped manner downward, bisecting the peduncle from above downward—that is, from the part where the aqueduct should be to the interpeduncular space, there remained only enough tissue in the anterior part to connect the two peduncles. The red nuclei on either side are deviated laterally, and the cells of both third nuclei in anteriorly are almost completely obliterated: only a few remain, which are diseased, while in its posterior portion, though fewer cells are destroyed, no normal cells can be found. The fibres of the posterior longitudinal bundle are replaced either by the tumour growth or deviated to one side. Some of the intra-medullary third nerve fibres are partially degenerated on either side, and there is complete destruction of all the fibres in the median raphe. The central grey matter surrounding the aqueduct is replaced by tumour cells, and there is also some degeneration of the fibres of the fourth nerve. The tumour in its anterior median part is free, growing into a cavity

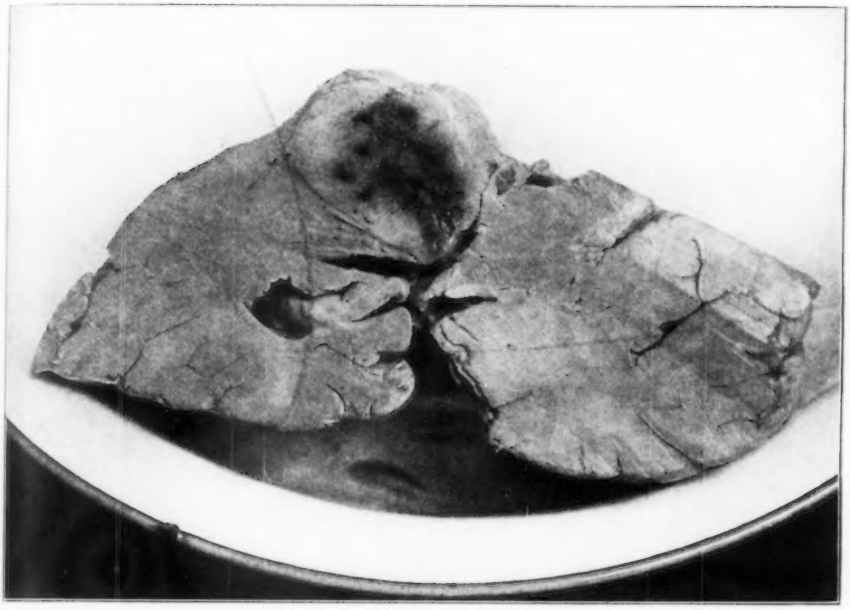

FIG. 2.

Case 1.—Vertical section showing outline of tumour, which is continuous with the right thalamus.

FIG. 3.

Case 1.—Photograph of cerebral peduncle, showing extension of tumour into the aqueduct of Sylvius; it grew ventrally, destroying the oculomotor nuclei and the commissural fibres, separating widely the red nuclei.

which has evidently been caused by it, but laterally there is an imperceptible merging, and tumour cells can be found in every part of the section. The foot of the cerebral peduncle, the lateral fillet, and the corpora quadrigemina are normal, although tumour cells can be found within these structures.

Sections made lower down in the cerebral peduncle and upper part of the pons show that the tumour has attacked the grey matter surrounding the aqueduct of Sylvius, irregularly infiltrating the surrounding structures, in the entire extent of the cerebral peduncle and the uppermost portion of the pons; the aqueduct with its ependymal cells is first seen flattened out with approximating walls at the region of the fourth nuclei. The cells of the fourth nuclei are few only in number, are much diseased, and are infiltrated and surrounded by tumour cells. Sections made at the level of the fifth nucleus show no trace of the tumour. No degeneration is found in the posterior longitudinal bundles, the lateral or median fillet, or in the motor nucleus. The medulla and cerebellum are normal.

The *cranial nerves*, with the exception of the second, third, and fourth, are normal. In the third nerves some of the axis cylinders are swollen, while in other areas they have disappeared. In places the myelin sheaths are greatly enlarged. The degeneration in the second nerve is not as great as that in the third.

CASE 2.—Reported in full, with DR. W. F. Guilfoyle, in the *Review of Neurology and Psychiatry*, June, 1910. A girl of 20, with a history of rheumatism, began gradually to lose vision. This was accompanied by flashes of light and double vision. Very soon afterwards she complained of headache, which was frontal and occipital, nausea, vomiting, and dizziness. She had several convulsions, which affected the left limbs, especially the arm, and during which time the eyes and head were deviated to the right. Soon afterwards she began to complain of pain all over her body, and especially of flashes of heat and prickly sensations in the legs, shoulders, and back, and, later, in the left arm. Vision became completely lost, choked disc developed on both sides, and was later followed by optic atrophy. Mercurial inunctions were of no value. About two months after the beginning of her symptoms she developed a slight ptosis of both eyeballs, more of the right, with unequal pupils, the reactions to light and movement being lost. There was marked exophthalmos of both eyeballs, more on the right, their axis being directed downward at an angle of 45 degrees. There was complete paralysis of associated ocular movement upwards, less to the right, still less to the left, and less so downwards. Convergence was lost in all directions, excepting in a downward median direction, and in this only an attempt was noticeable. The right motor fifth and tongue were paretic throughout. The lower portion of the right seventh nerve was weak. Hearing tests were unsatisfactory. The upper and lower limbs were weak and somewhat ataxic, although this was never satisfactorily demonstrated. All the reflexes were lost with the exception of the plantar. Sensation at no time was disturbed anywhere over the body. Incontinence of urine and fæces developed. The mentality of the patient gradually became worse. The

pains and flashes of heat were constantly complained of, and erythematous and acneiform eruptions, which were not persistent, appeared. Hallucinations of sight and hearing developed, and the ocular symptoms persisted to the end. There was no difficulty at any time in talking and swallowing, but she would at times yell without provocation. The patient had a constant tendency to roll the body and head to the right.

At the necropsy a vascular glioma was found in the left lateral ventricle extending into the third by means of the foramen of Monro. In its growth it pushed the structures before it, compressing the choroid plexus, the veins of Galen, the

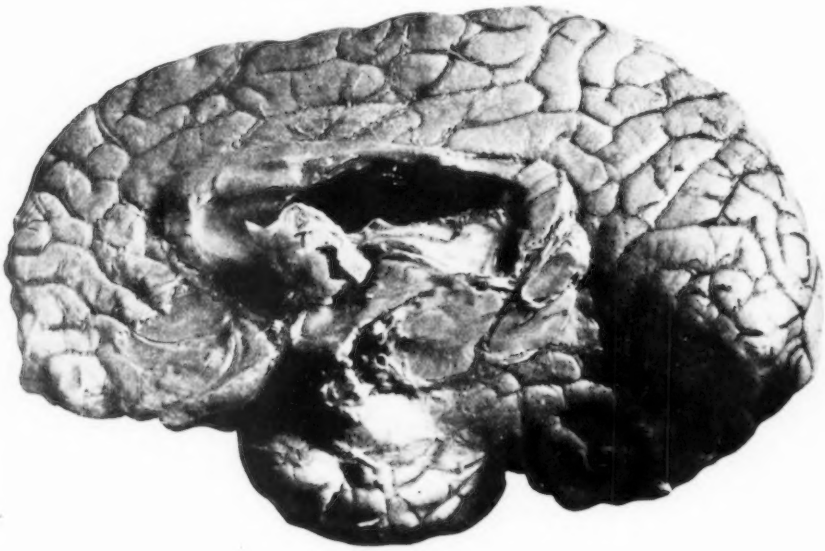

FIG. 4.

Case 3.—Colloid tumour (T), in the upper anterior portion of third ventricle compressing the foramina of Monro. Lateral ventricle much dilated. Third ventricle almost normal.

velum interpositum, and affected in part the left thalamus. In the third ventricle it occupied the upper anterior portion. The right thalamus was not at all affected, but the walls of the third ventricle were infiltrated with tumour tissue throughout. The cells of the third nuclei were distinctly diseased, but the posterior longitudinal bundles were not degenerated. All the ventricles were much dilated, as well as the aqueduct of Sylvius. There was degeneration of the posterior roots of the spinal cord.

CASE 3.—The history and material of this case were kindly furnished me by Dr. L. Webster Fox, the patient not having been seen by me during life.

E. P., at the time of death 27 years of age. The history was that in 1903 the left eye was injured, necessitating its removal. The patient was well after this for about three years, when he noticed that on bending either to the right or left he could not see well. At the same time he developed severe frontal headaches, nausea, and vomiting, which came on without apparent cause. With this he noticed that vision even in an erect posture in his remaining or right eye was not as good as it should be, and for this he consulted Dr. Fox. When examined he had a well-marked choked disc. From this time on the general symptoms of headache, nausea, and vomiting increased and vision became gradually poorer. The patient noticed, however, that if he held his body erect with his head tilted backward, vision was best, and that bending the head forward or to one side caused complete disappearance of sight, which would return on resuming an erect posture. The patient, in describing the return of vision, said "that he would first see a light-bluish point, then vision would gradually enlarge like an iris diaphragm until the whole field would come in."

Headache and nausea were less when his body was erect with head tilted back, and would also increase when bending forward or to one side. These were the only symptoms presented. Death occurred suddenly.

At necropsy a tumour about the size of a marble was found in the upper anterior portion of the third ventricle, it making a slight but equal groove for itself in both optic thalami. As is shown in Fig. 4, the growth was just anterior to the middle commissure, which was not distorted or pressed upon, and the foramina of Monro, which were dilated, were evidently closed up by the growth. The lateral ventricles were much dilated, the third ventricle being enlarged only a little. The aqueduct of Sylvius was normal. The tumour was almost round, whitish in appearance, and was easily displaced from its groove. It was loosely attached to the choroid plexus and in such position that it could very easily close up both foramina of Monro. It would also have been possible for it to be displaced from its groove by deviation of the head. Microscopic examination demonstrated a colloid cyst.

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CENTRAL NERVOUS RESPONSE TO PERIPHERAL NERVOUS DISTORTION.

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PART I.

IN 1900 Kennedy published the results of his experiments on cortical stimulation after nerve crossing [1]. Briefly stated, his experiments consisted in crossing the flexor and extensor nerves of the fore-paw of a dog, and then, after a suitable time-interval for regeneration, in stimulating the flexor centre in the cortex of the opposite side. Such stimulation produced extension, and not flexion, of the limb in question. Stimulation of the corresponding region in the opposite hemisphere produced normal flexion in the contralateral and normal fore-paw. These experiments of Kennedy have a direct bearing upon some work on the central nervous system upon which we have for some time been engaged. From many points of view it is a matter of considerable importance whether they are quite beyond the reach of critical attack. The only objection that could, we think, be urged against Kennedy's conclusions is that, as these flexion and extension centres in the dog are not widely separated from each other, and as the cortical configuration is very variable, a legitimate doubt may arise as to his having stimulated a flexion centre only and not included a portion of the extension centre through spread of the exciting current. It appeared to us, therefore, desirable to perform some experiments with the same end in view as Kennedy's, but with the operative interference such that a limb received its innervation, in whole or in part, from contralateral spinal roots. If, then, this limb showed movement on stimulation of the ipsilateral motor cortex, Kennedy's conclusions would be confirmed and no question could be raised as to the accidental stimulation of the contralateral cortex. The fore-limb of the dog was chosen, and the operation carried out on the brachial plexus. It was early discovered that to interchange the opposite cords

of the plexus, or even to cross mutually one of the main cords, was surgically impossible. We contented ourselves, therefore, with suturing the proximal end of one (the left) middle cord, cut as distal as possible, to the distal end of an opposite cord cut as proximal as possible.

As in Kennedy's experiments the time-intervals that elapsed between peripheral nerve regeneration and cortical stimulation were on the short side, we determined to allow a somewhat longer period for any educative process to be established in the central nervous system.

Methods.

The primary operation in each case was carried out under full anæsthesia produced by morphine injection, followed by chloroform-ether mixture. Ordinary aseptic and antiseptic precautions were taken, and the wound in each case healed by first intention. The nerves were sutured with chromicized gut, and irregular unions prevented by enclosing the unsutured proximal stump in rubber tubing. The dog was carefully tended in a special kennel. When regeneration was established, the animal was allowed a daily run in the dog-yard attached to the kennels. The second operation was carried out under anæsthesia produced by chloroform-ether mixture. The skull was trephined and the dura incised in the usual manner. Stimulation was carried out with ordinary double electrodes connected with the secondary coil of an inductorium. When the stimulation experiments, central and peripheral, were completed, the dog was killed by chloroform injection without any intermission of the anæsthesia having taken place.

Experimental.

Experiment I.—A female terrier, about 6 months old, was operated on. The *left* middle brachial cord was divided and its proximal stump brought under the trachea and sutured without tension to the distal end of the divided right middle cord. The proximal stump on the right side was drawn into a rubber tube and tied with silk to prevent regenerative sprouting. When the animal was examined, ten months later, co-ordination was found to be very good. The dog could walk and run naturally, and use either forepaw to hold down a bone. The stimulation experiment was carried out 308 days after the primary operation.

Stimulation in the right motor cortical area gave movement of both fore-limbs—namely, (*a*) extension of paw and flexion of elbow in the left (contralateral) fore-limb; (*b*) contraction of triceps and feeble contraction of paw extensors in the right (ipsilateral) fore-limb.

Stimulation in the left motor cortical area gave no movement of the ipsilateral fore-limb, but elicited various movements of the contralateral fore-limb, excluding contraction of triceps and paw extensors.

The peripheral nerves affected were exposed. The left middle brachial cord was found passing behind the trachea, and in regenerative union with the right middle cord. There were no irregular unions detectable, and the remains of the proximal stump of the right middle cord were found in the rubber tube, which was bleached but intact. The left middle cord was divided, and its distal end stimulated (of course proximal to the point of suture). Such stimulation gave movements in the right fore-limb only; the movements were confined to the triceps and paw extensors, and were similar in every respect to those elicited by ipsilateral cortical stimulation.

Experiment II.—A male terrier, about 9 months old, was operated on. Here an attempt was made to suture the left middle brachial cord to the whole of the right brachial plexus, but, as subsequent dissection showed, only the upper part of the right plexus was enclosed in the suture.

When examined nearly ten months afterwards the co-ordination was good. The stimulation experiment was carried out 301 days after the primary operation.

Stimulation in the right motor cortical area gave movement of both fore-limbs—namely, (*a*) headward movement of the limb, with strong extension in the left (contralateral) fore-limb; (*b*) elbow flexion and contraction of the shoulder muscles, particularly the deltoid, on the right side (ipsilateral).

Stimulation in the left motor cortical area gave no ipsilateral movement, but elicited movements of the contralateral fore-limb in which elbow flexion preponderated.

The peripheral nerves affected were exposed by dissection. The left middle cord was found passing behind the trachea. It was then cut proximal to the nerve suture and its distal end stimulated. Such stimulation gave no movement on the left side, but gave vigorous movement in the right fore-limb similar to that given by ipsilateral cortical stimulation.[1]

[1] In a third experiment the primary operation was similar to that in Experiment I. Unfortunately, in the stimulation experiment, six months later, the longitudinal sinus was opened and, owing, doubtless, to the severe hæmorrhage that ensued, we were unable to obtain movements of any limb in response to cortical stimulation.

Discussion of Results.

Our experiments, therefore, are in full accordance with those of Kennedy. They demonstrate (to use Kennedy's language) that an interchange of function can readily occur in the motor centres of the cerebral cortex. If attention be confined to the connexion between the cortex and the motor apparatus in the cord, we may state that here there is morphological fixity, but functional lability.

The older view that the pyramidal fibres make direct synaptic union with the motor cells of the ventral cornua is gradually being replaced by the idea that the pyramidal impulses enter the cord motor apparatus more on the sensory side. In the performance of a skilled movement certain reflexes must be inhibited, and this can be most economically performed, not by damping the individual motor neurons, but by throwing resistance into each reflex apparatus (or "draining" it) where there is greatest convergence of the appropriate reflex axes. But in the performance of an unrehearsed action it is also necessary to postulate that the cortex, if it does not play upon individual muscles, acts at least through the simplest co-ordinating mechanism—namely, that which produces a definite movement in a joint. This view is certainly borne out by Kennedy's and our experiments. Here the conducting paths remained unaltered from the cortex right down to the point of suture in the peripheral nerve-trunk, and yet co-ordination was acquired[1] [2].

We are justified, too, in assuming that in these experiments a considerable number of the sensory impressions from the affected limb, both integumental and proprioceptive, were projected on a wholly new cortical field, and yet these could apparently be used in guiding the discharges from the motor zone. Doubtless visual perceptions were used largely in the new co-ordination. In Kennedy's dogs, where flexor and extensor nerves were interchanged, visual guidance operated much in the same way as in the case of the microscopist who speedily learns to produce without conscious analysis a left-to-right movement of the magnified image by a right-to-left movement of the slide. In the dogs in our own experiments the whole sensory field had its normal cortical connexions except the two fore-limbs, one being fractionally apæsthetic. When a false movement is made the whole body participates and the stream of

[1] In Kennedy's case of union of the facial with the spinal accessory the lack of co-ordination, shown by the associated and synchronous movements of the shoulder and face, is easily explicable on the assumption of axon bifurcation. In this case the proximal spinal accessory was sutured to the distal spinal accessory and facial, giving the multiple path which we have shown to be favourable to axon-dichotomy.

afferents from retina, labyrinth, and body proprioceptors, after a number of repetitions, will compel the animal to abandon that particular motor grouping, normal as it may be at its cortical centre, and to become experimental in its movements. To assume that the new co-ordination is effected by known neurological mechanisms is surely safer than to postulate a supra-cortical control.

The expenses of this research were defrayed in part by a grant to one of us (W. A. O.) from the Science Committee of the British Medical Association, and in part by a grant to one of us (B. K.) from the Government Grant Committee of the Royal Society.

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Publications Recently Received.

[*Notes on a book under this heading do not preclude a subsequent review.*]

The Optic Nerve and the Accessory Sinuses of the Nose. By Professor A. Onodi. Authorized translation by J. Lückhoff, M.D., with 50 illustrations. Pp. 101. London: Baillière, Tindall and Cox, 1910.

The first sixty-eight pages of this careful monograph deal completely with the anatomical relations of the accessory sinuses of the nose to the optic nerve. All the varieties that may occur are carefully described and excellently illustrated. The second half is devoted to clinical facts bearing on the production of optic neuritis and atrophy from suppuration of the sinuses or injury to their walls by fractures or other lesions. The book treats a difficult subject with clearness and is based on first-hand observation. It is well translated and the illustrations are unusually good.

Three Lectures on Epilepsy, being the Morison Lectures delivered before the Royal College of Physicians of Edinburgh in 1910. By William Aldren Turner, M.D., F.R.C.P. Pp. 62. Edinburgh: Mackenzie, 1910.

The author devotes the first of these lectures to the manifestations of epilepsy, its predisposing causes, and the nature of the epileptic patient. After considering toxæmia as a cause of epilepsy, he passes on to a lecture on the border-line of the disease, and discusses the relation of epilepsy and hysteria, epileptoid phenomena, and such sleep symptoms as night terrors in the adult. The third lecture is devoted to treatment, and the advice given is succinct and excellent. This work can be recommended to all practitioners anxious to obtain in small compass a knowledge of the main facts of epilepsy and its treatment.

L'Aphasie dans ses rapports avec la démence et les vésanies. Avec cinq planches et quatre figures dans le texte. Pp. 253. Paris: Steinheil, 1910.

The greater part of this monograph is devoted to a consideration of the mental state of patients with aphasia. Marie holds that every case of aphasia shows diminished intelligence, though this diminution in no way corresponds

with a dementia. The author states that in some cases the intelligence is untouched, but that some cases of aphasia are true organic dements. This dementia is due to lesions other than that causing the aphasia. Fifty-two cases are considered in varying detail.

Nouveau Traité de Médecine et de Thérapeutique. [Gilbert et Thoinot.] XXI Sémiologie Nerveuse. Par Achard, Marie, Gilbert Ballet, Lévi, Léri, Laignel-Lavartine. Avec 129 figures intercalées dans le texte. Pp. 629. Paris: Baillière, 1910.

This volume of the "Traité de Médecine" deals with grouped signs of nervous disease. Thus, apoplexy, aphasia, hemiplegia, paraplegia, delirium, convulsions are treated in separate chapters by different writers. Contracture, tremor, vertigo, headache, trophic and secretory troubles are all fully dealt with. Such an arrangement is unusual in English text-books, but is excellently carried out. The question of the causes of aphasia is discussed with admirable clearness and completeness. The volume is well illustrated, and the only fault in arrangement is the absence of an index.

Surgery of the Brain and Spinal Cord, based on Personal Experiences. By Professor Fedor Krause. Translated by Professor Herman A. Haubold. Vol. I, with 63 figures in the text, 24 coloured plates, and one half-tone plate. Pp. 282. London: H. K. Lewis, 1910.

This is a translation of the first part of Professor Krause's admirable book on brain surgery, a work of great interest from the fact that it is a record of personal experience. All the principal methods of attacking various portions of the brain are given, and are illustrated with lithographic plates. The English is slipshod and careless.

Organic and Functional Nervous Diseases: a Text-book of Neurology. By M. Allen Starr, M.D., &c. Third edition, thoroughly revised. Illustrated with 300 engravings in the text and 29 plates. Pp. 911. London: Baillière, Tindall and Cox, 1910.

This text-book is already well known, and much new matter has been added in the present edition. It is fully illustrated, but some of the photographs of the spinal cord might be lettered for the sake of the student unfamiliar with spinal topography. The results of the author's own researches are given clearly and fully, especially in the chapter on Spinal Segmentation.

Writers of "Original Articles and Clinical Cases" are supplied free of charge with 50 copies reprinted in the form in which the paper stands in the pages of "Brain." If reprints are required in pamphlet form, with wrapper, title-page, &c., and re-numbered pages, they must be ordered, at the expense of the writers, from Messrs. BALE, SONS & DANIELSSON, Ltd., 83-91, Great Titchfield Street, London, W.

Members of the Neurological Section of the Royal Society of Medicine can obtain the Index of "Brain" for the Volumes I. to XXIII. inclusive, that is, from its commencement to the end of 1900, from Messrs. MACMILLAN & CO., Ltd., St. Martin's Street, London, W.C., at the price of 6s. 6d., post free.

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EDITOR.